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THE PARANASAL SINUSES. SUMMARY OF LISTINGS IN THE INDEX MEDICUS FOR 1960.

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Part II.

(Continued from October Issue.)

Miodonski[113] suggests that when operating on a large frontal sinus one may prevent the closing off of the lateral recess by placing a finger-cot into the sinus which is drawn all the way to the external angle by means of a heavy thread which anchors it there.

Chardot and Carolus[114] describe several autoplastic procedures they have employed for the immediate closure of defects following removal of malignant tumors of the orbit and orbito-sinus areas. Nearly all of them originated in the orbit or its soft tissue coverings with secondary invasion of the sinuses of lesser degree.

Vizzoni[115] claims excellent results in the postoperative treatment of the antrum following the Caldwell Luc or De Lima operations. At the conclusion of the operation the cavity is dried thoroughly by means of a gauze pack. When all of the bleeding has been controlled he insufflates some sulfa powder and repacks with plain or iodoform gauze. After 48 hours this is removed, and again all clots and dead tissue are removed, the cavity irrigated and repacked. This procedure is repeated daily for about five days. The sublabial wound is then sutured, and the cavity remains free. The point is that

this procedure prevents the formation of clots in the cavity and controls the development of the granulations preventing adhesions which predispose to the formation of isolated pockets, all of which facilitates reinfection.

Belfort[116] prefers a vertical or oblique incision for exposing the antrum wall because it avoids cutting the fibers of the infraorbital nerve, and subsequent anesthesia of the cheek and teeth. He also takes great care in making the counter opening into the nose smooth and rounded and as close to the floor as possible.

Multanen[117] has successfully closed several large antro-alveolar fistulas by means of two flaps. It is similar to the one described by Hersch, except that the flaps are reversed. The buccal flap being the thinner, is reflected over the opening and sutured in place and then covered by a transposed palatal flap which is nourished by the ascending palatal artery and is thicker than the other. The absence of concurrent sinus infection may have been a factor in the author's success with the four cases he cited.

Burch and Funk[118] report a case of oroantral fistula in a 44-year-old male, which resulted from extraction of a second molar several years before and had once been unsuccessfully operated upon. Because of the size of the bony fistula it was deemed inadvisable to attempt to close it. Instead, the opening was made a permanent one by turning the mucoperiosteum over the bony margin fixed by several sutures carried through the antral wall, thus affording a smooth soft lining to support a prosthesis which was subsequently employed.

Fisher and Anthony[119] have performed more than 500 intranasal antrostomies in a period of 14 years with satisfying results. It is indicated in cases of chronic maxillary sinusitis, recurrent acute attacks, allergic sinusitis complicated by infection, fractures of the orbit, offering facilities from manipulation through the opening and chronic draining fistulas. They find it possible to perform the operation under local even in children as young as 12 years. The operative technique is given in detail.

NONSURGICAL THERAPY.

Satisfactory results with the use of various antihistaminics and vasoconstrictors are reported by a number of observers. Edmunds[120] used Dimetane for vasomotor rhinitis and allergy in 200 cases at a U. S. Naval Hospital with apparent cure in 57 and great improvement in 89.

Schwartz and Slasman[121] found Nolamine useful in 35 cases. No side effects were noted. A similar report by Villanyi and Stillwater[122] using Rynatan in 17 adults and 18 children notes no ill effects.

Kluger[123] used Otravin in 200 cases and found considerable relief from nasal congestion without side effects; however, the drug was less effective in the true allergic patients. In such cases he obtained good results from the injection of Hyaluronidase into the turbinals. When this is followed by the injection of Hydrocortisone or Prednisone it is particularly effective in relieving pre-menstrual engorgement of the turbinals.

MacLaurin[124] obtained satisfactory results from the use of Triaminic HC in 89 per cent of his cases. In cases where following improvement with Triaminic HC the patients were given Triaminic alone, one-third of the cases relapsed. He concludes, therefore, that two-thirds of his cases maintained their initial improvement even when the HC was eliminated.

In another contribution by MacLaurin *et al.*[125] the effects of various orally administrated sympathomimetics were studied by observing the subjective responses, degree of shrinkage, blood pressure and side effects. A placebo was used in some cases, and it was interesting to note that the blood pressure was elevated in many of these (20 mm. or over). It was shown that ephedrin produced a greater vasoconstriction than any of the other drugs as demonstrated by rhinometric readings.

Despons and Le Mouel[126] claim that the danger of lipoid pneumonia from the use of oily solutions in the nose can be avoided if the medium employed is a neutralized vegetable

oil. They have shown also by animal experiments that such solutions do not damage either the epithelium or its substructure, although there may be a temporary slowing of the ciliary beat. If used judiciously by fine atomizing or by aerosol medicaments dissolved in these oils, they remain in contact with the mucosa for a much longer time than aqueous solutions thus prolonging their therapeutic effects.

The article is a lengthy one covering the literature on lipoid pneumonia and the local effects of oily solutions.

Eagle[127] found that streptokinase and streptodomase were very effective in resolving post-traumatic and postoperative edema, blood clots and infection. They employed them in tablet form (varidose), the tablets being held between the cheek and alveolus to undergo slow absorption through the buccal mucosa. He demonstrated its value in cases of reaction following a Caldwell-Luc operation, frontal sinus surgery and some cases of serous otitis media. The tablets must not be swallowed since they have no benefit when they reach the stomach.

Silbert[128] has found that chronic bronchial disease is frequently the result of a sinus and allergic condition in childhood which was not checked. He has been particularly interested in the result obtained in treating these conditions with an enzyme (trypsin). This is given in conjunction with specific therapy as indicated in the allergic cases.

Reversible changes, as shown in successive X-ray pictures, indicate that the therapy is frequently of lasting benefit. His main point is that it is never too early to diagnose and treat sinus disease regardless of the age of the patient.

Tyrrel *et al.*[129] inoculated 18 volunteers intranasally with various doses of a strain of parainfluenza 1 virus. Nine became infected, and six of these developed illnesses resembling the common cold.

Twenty volunteers received parainfluenza 3 virus. Eight became infected, of whom six developed cold-like illnesses; one had an illness like influenza, and one remained well. The

colds produced were clinically indistinguishable from those occurring after instillation of washings containing a cold virus, but the incubation period was longer in the parainfluenza virus infections.

Mikhailovskii (English Abstract)[130]: "The negative aspects of antibiotic therapy in otolaryngology are conditioned by their untoward reactions. They are to be employed only in strict indications thereof and in appropriate doses, paying due consideration to the action of the antibiotic on the given flora, the reaction of the body to its administration, and the technique of the introduction. Appropriate prophylactic measures and complex treatment of the patient are of importance."

Koch[131] discusses the use of antibiotic therapy in minor affections of the respiratory tract and points out the failure in many cases due to the following factors:

1. Therapy with antibiotics in diseases that are not bacterial in origin.

2. Incorrect application of the therapy.

3. Inadequate dosage.

4. Too early discontinuance of the therapy.

The correct diagnosis of etiologic factors and clinical laboratory findings are of prime importance.

Specifically in a truly infectious rhinitis and beginning sinusitis the author has had good results from the use of a spray combining a corticosteroid such as Prednisolone with the antibiotic "Framycetin" and a vasoconstrictor, all of which is packaged under the name of "Sofrasolon".

Antibiotics by injection or orally are indicated only if there are symptoms suggesting systemic involvement.

CLINICAL.

This chapter contains a wide variety of interesting clinical reports of some very unusual conditions.

A symposium on sinus conditions fills the greater part of one issue of *Montpelier Medical*.[132] The articles include one by J. Terracol, "Le canal naso-frontal," "Les liaisons des cavites sinuso-nasales" by Terracol, Guerrier and Ardouin, "La flore microbienne dans les sinusitis" by Terracol, "Les meningites d'origine naso-sinusienne" by Terracol and Piquet and "Les tumeurs benignes des sinus" by Terracol and Guerrier.

These are all in the nature of a review with liberal citations from the literature and may have some value from the "summing up" standpoint. The articles on the nasofrontal duct, meningitis and benign tumors are perhaps more thorough than one may find in standard textbooks. They are certainly too detailed for adequate abstracting.

Juni[133] states that three basic pathologic mechanisms account for disease in the sinuses: vasomotor phenomena including allergy, infection and tumors. The author favors the use of autogenous vaccines and physiologic rather than surgical management in most cases. A wiped smear technique for diagnosis is of great value, and when surgery is indicated the obliterative technique with fat implants is highly successful.

Formby[134] reviews the history of the maxillary sinus and credits Leonardo da Vinci as being the first to describe it accurately. The paper which is the President's address covers most of the well known facts about the embryology, pathology, etc., of the sinus. On the subject of therapy, he advises conservatism wherever possible, withholding surgery until other measures have been tried and found to be ineffective.

Forbes and Bradley[135] had a case of extensive bilateral polypi in a man of 70 who was operated upon via the intranasal route, which terminated fatally from a purulent meningitis. On the day following surgery he collapsed, became unconscious and passed away within 48 hours. Autopsy revealed a large gap in the cribriform area of the base of the skull affording a free communication between the nasal cavities, the antrums and the endocranium. The dura was lacerated, and pus and blood were widely distributed.

The authors point to the chronicity of the process in cases of this kind as a factor in causing absorption of intervening bone and claim it is to the credit of good otolaryngologists that these accidents don't occur more often.

Mundnich[136] discusses seven cases reported in recent years where serious complications including several deaths followed antrum puncture. Aside from the cases where the needle point had been imbedded in swollen mucosa or had delivered air into the veins beneath the stripped mucosa, there were several in which serious symptoms developed when the needle point was free in the cavity. In addition to the cases of air embolism he directs attention to those in which fluid or air has been injected into the orbital cavity or the tissues of the cheek. A medico-legal problem is involved in cases where the puncture results in a fatality. This leads the author into a discussion of the necessary precautions one must take to avoid complications. Failure to observe the rules he lays down renders the surgeon liable. According to the author, the Lichwitz needle should have a laterally placed opening; when introduced one should first aspirate. Should neither air nor fluid appear, one may cautiously inject a few cc. of saline and repeat the procedure. If patency is then not apparent, the procedure should be halted. If a contrast medium is used it should be preceded by an irrigation and only a water soluble mixture used for contrast studies.

He claims that irrigation through the natural opening is not without risk since cases of complications have been reported; however, this point is rejected by Spann and Jacobi who assert that in their experience the middle meatus approach with a blunt cannula is far safer than inferior meatus needle puncture. They state that this procedure requires delicacy of touch and long practice. When one becomes adept at it, the dangers of complication are minimized almost to zero.

Responding to these comments Mundnich stated that inasmuch as the vast majority of antrum irrigations done in Germany are performed through needle puncture the results cannot be compared with the middle meatus procedure be-

cause of this disparity in numbers. He points out also that in many cases the cannula does not enter the natural opening but passes through the membranous portion of the wall or through an accessory opening.

Finally all of the essayists seemed to agree that air should never be forced through needle or the cannula.

Murphy, Wilmer and Claiborne[137] report a case of heart block in a woman of 54 which recurred while a stream of saline solution was directed into the area of the sphenoid sinus. Cyanosis and loss of consciousness ensued, the blood pressure dropped to zero, and there was a brief convulsive seizure. The patient was removed to the hospital where she remained for two weeks during which time therapy was directed to overcoming the cerebral and myocardial damage. There was a history of a prior heart attack, and the assumption was that the incidence was the result of vagal stimulation via a trigemino vagal reflex. The electrocardiograms taken before, during and after the episode demonstrate the course of the myocardial involvement.

Horsfall[138] lists the viral diseases of the respiratory tract as the following—common cold, pharyngitis, herpangina, pleurodynia, acute respiratory disease, laryngo-tracheo-bronchitis, influenza, and primary atypical pneumonia. Along with these he names the offenders and the immunologic types. Viral diseases are less amenable to therapy because of the wide diversity of their structures and size; yet all viruses are spheroidal, and their most important components are protein and nucleic acid. These are shown in a drawing depicting the structure of a few of the better known viruses. The author describes how the virus penetrates the animal cell, namely through its disintegration, leaving only the nucleic acid particle as the offending agent. Once the nucleic acid has entered the cell it reproduces new virus particles in conjunction with the cell materials. This synthesis takes place within hours. Large numbers of these new viruses then escape in a stream from the infected cell. To combat the propagation of the virus a number of agents have been suggested which are effective in certain types of infection,

but only to a limited degree. The problem is to evolve an agent that will halt the cell changes which occur when the virus has entered it. Increasing knowledge of the viral processes within the cell may lead to clearer understanding of it and possibly to the proper means of combating it.

Andrews and Kneeland[139] also offer some comments on the subject of virus infections and the common cold. The latter states that passive immunity in the newborn wanes with the passage of time, when contact with an array of respiratory viruses builds up a pattern of active immunity against agents which are antigenically patent. Most of these contrive to protect him into adult life, against severe viral diseases until some new agent of high infectivity develops such as Asian Flu.

Williams *et al.*[140] investigated the incidence of Staphylococcus carriers in four different hospitals covering over 1,000 patients. Nasal swabs taken within the first few days after admission to a male surgical ward yielded 38 per cent carriers. Another 13 per cent had other penicillin resistant strains. Twenty-five were swabbed after eight weeks in the ward and 68 per cent were carriers plus 52 per cent with other resistant organisms. Non-carriers on admission acquired resistant strains more readily than those initially carrying sensitive strains. Sixty-four out of 165 patients treated with antibiotics acquired penicillin resistant strains; 39 acquired tetracycline resistant strains.

The incidence of postoperative staphylococcic wound sepsis was 2 per cent in 342 patients who were never nasal carriers and 7.1 per cent of the 380 who were.

Out of a total of 1,319 patients 4.5 per cent developed sepsis elsewhere than in a wound.

This study points to the importance of reducing the acquisition of staphylococci in the nose.

Satyanarayana[141] presents an excellent description of rhinosporidiosis based on observation of 255 cases. He describes the life history of the parasites, the clinical characteristics of the disease process, mode of infection, pathology, etc. The

lesions appear on the mucous membrane as small red granulations which increase in bulk and become pedunculated in lobular masses. They are vascular and bleed readily. Examination of the mass with a hand lens will disclose numerous white spots which represent the bulging sporangia giving the mass a strawberry-like appearance. The article is replete with illustrations of typical cases, photomicrographs of the lesions and pictures of the spores in various stages of development.

The disease must be very common in India. In this country it is very rare. It is more common in agricultural areas, and apparently the spores may abound in streams, in sand and possibly among domestic animals.

Diagnosis is easy if an impression smear from the growth mixed with a drop of water is pressed between a slide and cover glass. If stained with a Romanowsky stain the spores containing characteristic spherules are readily demonstrated.

Vervoorn and DeJager[142] report a case of rhinoscleroma in a 29-year-old male of Ghana. The nares were blocked by masses of granulomatous tissue which was friable and hemorrhagic. Histologic examination of the tissue showed masses of plasma and Mikulicz's cells, the latter containing bacteria and also mast cells. Recurrence of the growth was noted after primary removal, but yielded finally after the administration of streptomycin in daily doses of 1 gm.

Postnov (English summary)[143]: "A case of rhinoscleroma is described, associated with local amyloidosis in the skin of the upper lip and the nasal mucosa in a 50-year-old man. The combination of rhinoscleroma with local amyloidosis changes considerably the picture of tissue alterations characteristic of this inflammation, particularly by the appearance of granulomas of the foreign bodies. Its diagnosis in these conditions is rather difficult. In conjunction with this a suggestion is made that rhinoscleroma plays an etiological role in the development of different forms of local laryngeal amyloidosis."

According to Cody and Hallberg[144] there has never been a

case of pyocele of the sphenoid reported in the literature, although nine cases of mucocele have been listed. The case they report was in a woman of 58 who had suffered from supraorbital and occipital headaches for a long time and was also a victim of hypertension. For several years she had been aware of a foul odor from the nose. Probing and suction of the sinus yielded some cheesy material. X-rays showed a cloudy sphenoid. The sinus was opened by enlarging the natural ostium and was found to be filled with foul smelling cheesy detritus and lined by thickened mucosa. Cultures revealed Hemophilus influenza and Staphylococcus. Improvement followed, and the general condition improved by the use of vascular depressants with complete recovery from the headaches.

Felty's syndrome is described as a combination of chronic rheumatoid arthritis, splenomegaly and leukopenia. In the case reported by Machab Jones[145] a 50-year-old man suffering from a series of colds had a tooth removed which was followed by spontaneous extrusion of an adjacent molar and a swelling of the hard palate. He was found to have a very large spleen and white count of 1,300 with 30 per cent neutrophils. A generalized rheumatoid arthritis was also diagnosed. Splenectomy was followed by prompt rise in the white count to normal with normal percentage of neutrophils.

Weller, Joseph and Hora[146] report a case of involvement of the maxillary and ethmoid sinuses, the orbit and adjacent structures by Aspergillus flavus in a colored male of 31. The case was followed for nearly four years during which time several operations were performed on the antrum, orbit and temporal fossa for the removal of granulomatous tissue. Remissions followed intensive therapy with intravenous injections of amphotericin B (Fungizone) and local insufflations of powdered Nystatin. Reaction to the intravenous therapy consisted of fever, headache and nausea which could be ameliorated by aspirin, phenacetin and codein. Although the process seemed to be halted, the patient was not definitely cured, since the last tissue examination revealed the fungus still present.

A case of cholesteatoma of the frontal sinus of traumatic origin is described by Birnmeyer.[147] A woman of 30 noticed that her right eye was "swelling" the past six months. The bulb was proptosed but mobile—there was no pain. Nasal examination negative. X-rays showed a marked distention of the frontal sinus with erosion of the orbital roof. Frontal sinus irrigation returned clear. At operation a large cholesteatoma was uncovered extending deeply and laterally. The mesial portion of the sinus was clear. When the mass was removed, a scar was noted which was bound to the skin overlying the temporal aspect of the bone. Here a bony defect measuring 2 x 20 mm. was encountered. This lesion was traced back to a fall against a sharp object. The area was excised, and primary healing ensued. The author stresses the importance of careful attention to wounds involving the facial bones.

Grunberg[148] reports a case of aspergillosis of the antrum in a woman of 57. There was nothing characteristic in the symptomatology or local findings. Headaches and facial pains were constant. X-ray pictures showed thickening of the antrum lining and numerous pin point shadows scattered throughout. When opened, the sinus was found to contain a moderate amount of brownish black material which showed mycelia, spores and "fruchttrager".

Antibiotics were not employed in this case, and the author states that thus far their value in these cases is uncertain. Heretofore the therapy has consisted of the administration of iodides and the local use of various dyes.

Esteban[149] calls attention to endocrinologic disorders as basic causes for the development of allergic involvement of the nose and sinuses. Disequilibrium of the neurovegetative system, hypothyroidism and imbalance in the electrolytes have been found in association with sinus pathology. He cites one case where persistent headaches with cloudy sinuses showed a small pituitary in the X-ray pictures, a low metabolic rate and a hypercholesteremia. Relief followed the administration of thyroid extract.

Wodak[150] shows some nice color photos of a case of herpes involving the second and third branches of the trigeminus and the glossopharyngeus. Most interesting are the pictures of the interior of the antrum taken through the antroscope which shows the typical herpetic lesions in the mucosa.

Considering the sinus complications of dental origin, Vigneul[151] has no difficulty when discussing such obvious conditions as dentigerous cysts and oroantral fistula. He runs into trouble, however, when he takes up the possible connection between dental infection and sinus infection and particularly in analyzing the symptom of pain. The most that can be said is that the conditions are unilateral, and much depends upon the skill and experience of the dental surgeon in analyzing his X-ray pictures of the teeth and the alveolus.

Krasne[152] lays considerable stress on the psychological implications in histaminic cephalalgia. These include a disposition to sustained emotional states, compulsive behavior, insecurity, lack of self-confidence and over conscientious striving for perfection. He discusses the differential diagnosis between this type of headache and similar disorders such as trigeminal neuralgia, migraine and nasal sinusitis.

Reinhold and Sauerbrey[153] relate that the X-ray pictures of 740 patients complaining of headaches revealed evidence of sinus disease in 33.1 per cent; however, the authors fail to show any connection between the sinuses and the headaches inasmuch as there is no mention of the pathology present or whether therapy was employed and if so, what the results were. In a number of the cases they found osteochondrosis of the cervical vertebra and also quite a few sharply deviated septums. Other causes noted were hyperostosis frontalis and tumors of the pituitary.

Thomas,[154] reviewing a large series of private patients complaining of headaches, facial neuralgias and recurring upper respiratory infections, has found a considerable percentage of them to be due to latent or previously undiagnosed sinusitis. The condition can be diagnosed by diligent investi-

gation of the sinuses, by irrigation, by the use of tampons of 5 per cent Ichthyol in glycerin, resection of the septum when indicated, and autogenous vaccines.

TRAUMA—FOREIGN BODIES.

The danger of late meningitis following trauma involving the sinuses is well exemplified in the following group of abstracts.

Powiertowski, Malecki and Buttner[155] suggest a two-stage operation for the treatment of skull fractures involving the sinuses under certain conditions. These include cases of laceration of the dura, even though cerebrospinal rhinorrhea may not be present. This may happen in certain closed cases; however, if the X-rays show a fracture of the sinus roof and there are no signs of sinus infection, the intracranial approach alone should be employed; otherwise they advise a rhinologic procedure to exenterate the infected sinus followed by the intracranial procedure for the closure of the dural rent. If X-rays reveal a simple linear fracture uncomplicated by rhinorrhea or pneumatocele, the intracranial approach may be dispensed with. If the sinuses are destroyed by the accident and there are no radiological signs of fracture of the sinus roof, only the rhinological operation need be done.

The combined operations are advised in all patients with fractures of the sinus roof causing laceration of the dura with simultaneous destruction of the sinus. It is indicated also when there is a history of long standing sinusitis. Every case with rhinorrhea should also be operated intracranially. The authors have operated on 90 patients, 48 of whom had had previous operations. Their mortality rate was less than 5 per cent.

Laskiewicz[156] describes in detail the history and course of 14 cases of trauma and foreign bodies involving the sinuses. In most cases he advises thorough radiological study and wide exposure of the frontal area, especially in severe cases, by reflecting a skin periosteal flap starting at the hair line and based on the supraorbital border so as to expose the

entire frontal bones. Bone fragments are removed and lacerations of the dura sutured or covered by adjacent periosteum or fascia lata. He cites several authors who prefer using fibrin sponge material. If the frontal sinuses are small, they may be obliterated by applying the skin periosteum to the dura. In the case of largely pneumatized frontals, he suggests exenteration of the ethmoids, maintaining a wide passageway to the frontal. The article is well illustrated.

Csillag[157] reports a case of a young lad sustaining a blow over the forehead by an unshod colt and being in a somnolent state and vomiting when seen a few hours later. The lids were swollen and discolored and two wounds noted—one over the root of the nose which was bashed in and an open wound over the forehead from which greyish brain matter extruded. X-rays showed a fracture of the outer table extending y-shaped upward toward the midline and backward to the parietal area. An incision was made uniting the two wounds, numerous fragments of bone which had penetrated the dura were removed. The laceration was sutured. The sunken nasal bridge was lifted up and held by intranasal packing and the wound closed with drains. Antibiotics were given and after 50 days the boy was discharged with the admonition to return at intervals for examination because of the possibility of late complications. This was not done, but the boy was brought in about a year later with a meningitis which recurred twice within a few months. Pneumococci were isolated, and the author speculates on the question as to whether the meningitis was independent of the fracture, although the general opinion is held that late meningitis through the original portal of entry, namely the fracture, is more probable.

Correll[158] demonstrates again the importance of thorough investigation of the dura in all cases of frontocranial trauma where a fracture through the frontal sinus posterior wall is demonstrated or suspected. At the age of ten, a little girl sustained a compound comminuted fracture in the frontal region involving the sinus. There was every evidence of intracranial involvement, but no surgery was attempted at the time. Subsequently, the patient complained of intermit-

tent headaches and a prominence over the brow which at times became red and swollen. When admitted at the age of 32, the X-rays showed a translucent area in the frontal sinus with some bone destruction and evidence of an old fracture near the frontal notch. The area was explored through the frontal sinus which was diseased, and a mucocele was found which had eroded the anterior, posterior and medial walls. The posterior portion of the sac which was adherent to the dura was left in place. Within 24 hours meningeal symptoms appeared, and when the meningitis developed, a second operation was performed, namely a frontal craniotomy. This disclosed granulation tissue extending from the sinus through a large dural defect into the brain which was necrotic. After thorough exenteration of the diseased area and proper treatment with antibiotics, the dural defect was closed by means of a pedicled graft of pericrania with some frontalis muscle, shutting it off from the sinus which was drained through the nose. Recovery was complete.

Neuss,[159] analyzing reports in the literature covering cases of late meningitis following skull fractures, found that the incidence in the cases of sinus involvement was no greater than in the group involving the auditory apparatus. The prognosis, therefore, is no different in the two instances. It is of interest to note that the latent period on the average was about the same, namely 4.06 vs. 4.12 years. These figures were derived from a group of 65 cases affecting the sinus area and 41 cases involving the ear.

Applebaum[160] reviewed a series of 91 cases of meningitis following trauma to the head and face. In only 48 of these was there definite evidence of skull fracture, yet the symptoms might indicate that in many others, such a lesion was present though not demonstrable. The importance of bleeding from the ears or the nose or the presence of rhinorrhea or otorrhea as an indication of laceration of the dura, is stressed. The author presents a number of tables showing the types of trauma, the interval between trauma and appearance of meningitis, age distribution, principle symptoms, causative organisms and final outcome. The percentage of

recoveries has risen in recent years due to the influence of antibiotics.

Hager[161] states that prompt sealing off of dural defects following fronto-basilar trauma is imperative if one is to prevent nasal infection from passing into the endocranial tissues; however, if a meningitis is already present, the question of closing the defect may be difficult to decide. In eight of such cases the author did close the defect despite evidence of endocranial infection with good results. He claims that individual circumstances in each case determine the feasibility of closure as against temporizing. If the dural inflammation is of recent onset showing retention of good vitality and the meninges are not too badly inflamed, the dura may be closed safely as long as the antibiotic therapy is adequate. In the four cases where closure of the dura was deferred, the reasons given were presence of infection in the sinuses and a dural tear in an area not readily accessible. In these cases the defect was covered by a form of gelfoam.

Frackelton[162] outlines briefly the essential points in the care of injuries to facial structures sustained in athletic engagements. He covers the familiar ground only on the essentials of early management. Detailed techniques require a much larger dissertation.

Von Gralath[163] in a rather lengthy and profusely illustrated article demonstrates various procedures for the reduction of facial fractures. Most of them conform to the techniques we find in standard textbooks on the subject. The author points out the importance of achieving perfect alignment of the teeth and shows the results of muscle pull depending upon the location of the fracture.

Lund Iverson[164] reporting on the results in 71 zygomatico maxillary fractures states that 68 were treated by open reduction through the usual Caldwell Luc incision. In 42.5 per cent simple reduction without fixation was effective. In 13 per cent wire sutures were necessary, in 9 per cent plastic support and wire fixation and in 4.5 per cent fixation with arch bar and splint.

Holczabek[165] reported a case where a lad died as a result of a beating in a fight showing only minor facial lesions. Autopsy revealed marked intracranial damage. Hemorrhages into the sinuses were noted, and the author considers this a significant finding.

Novotnyo[167] tells of an 11-year-old boy who, while playing with some friends shooting home made bow and arrows, was accidently struck by an arrow causing a wound just below the inner corner of the eye. Examination failed to reveal any damage to the eye. The wound healed spontaneously but soon broke open as a discharging fistula. X-rays showed clouding of the ethmoids and antrum. The area was explored through the fistula which led through the lamina papyracea into the ethmoid labyrinth from which a piece of wood measuring 3 x .75 cm. was extracted.

Brain[168] reports a case of rhinolithiasis in a woman of 59. Three calcareous deposits were removed from the antrum which contained purulent material. The subjective symptoms were persistent nasal obstruction and infraorbital pain. Puncture irrigation failed because of obstruction. X-rays revealed the isolated dark shadows in the sinus. Analysis of the stones showed 10 per cent organic and 90 per cent inorganic matter. The author presents brief abstracts of 13 similar cases reported in the literature. In the absence of foreign bodies introduced from without, it is assumed that the stones were formed from the products of chronic suppuration.

Localization and removal of metallic foreign bodies from the environment of the sinuses may be very difficult in many cases. The author, Jatho,[169] mentions the Berman locater as described by Von Tornquist as satisfactory but too expensive.

Instead he describes a simple apparatus capable of delivering a smooth galvanic current of low voltage which is carried into the tissues by a long needle. The circuit is completed through a moist pad placed around the arm. The procedure is first to determine the location of the F. B. by means of X-ray pictures A . P as well as lateral and at times planagraphic views. When the area is exposed the needle

electrode is introduced, and the gauge recording will show an increase in the voltage as the needle approaches the F. B. This procedure has been successful in localizing F. B. as small as 4 mm.

Kotyza[170] reports two cases of foreign bodies in the sinuses. One was due to clone operation in which bone fragments were left behind. The other was in a five-year-old boy who fell from a tree, sustaining a laceration of the lids. Secondary infection led to reoperation where the infected ethmoids were found to contain three fairly large fragments of twigs and a number of smaller pieces. The author points to the failure of X-rays to disclose such non-opaque bodies.

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NINTH CONGRESS OF THE PAN-PACIFIC SURGICAL ASSOCIATION, OTOLARYNGOLOGICAL SECTION.

The Ninth Congress of the Pan-Pacific Surgical Association Section on Otolaryngology will be held in Honolulu, Hawaii, November 5-13, 1963.

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OTOSCLEROSIS OF THE STAPES.*†

A Study of the Lesion by Histochemical Procedures and Fluorescence Microscopy.

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INTRODUCTION.

Many significant contributions to the histopathology of otosclerosis appeared over a period of years prior to Lempert's fenestration operation. These observations were usually made on temporal bone sections for which the older methods of fixation, decalcification, embedding and staining were utilized. Although the pathology of the lesions became well established, the etiology of the disease remains obscure.

With the fenestration operation it was possible occasionally to obtain a small piece of bone involved with otosclerosis. The specimens were scarce, otherwise it would have offered an opportunity for study of the lesion by other methods that were not feasible on sections of the temporal bone or autopsy material that was unsuitable for use. The stapes replacement procedures have provided not only an opportunity to procure otosclerotic bone in sufficient amounts but also in a fresh state required for special histochemical techniques. Histopathological observations on this type of material by Henner, Guilford, Shea and Jeantet[1] have revealed its excellent suitability for studies by newer methods.

The present study is concerned with the histochemistry of the otosclerotic lesion present in the stapes removed at the

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time of surgery. Particular attention has been devoted to the nature of the intercellular substance, cellular content and evidence of activity in the otosclerotic bone; however, the results are to be considered as preliminary because of the lack of nonotosclerotic stapes for controls and difficulties encountered in attempting to apply some of the highly specialized methods to this type of material.

MATERIAL AND METHODS.

The material for the present experiments was comprised of 110 specimens of head, crura and footplate of stapes removed during stapes replacement operations. The specimens were prepared for study by a variety of fixatives and staining procedures.

1. Mucopolysaccharides. Forty-five specimens were fixed immediately after removal in 10 per cent neutral formalin, cold 80 per cent alcohol or acetone. After 24 hours in the fixative solution they were decalcified in a sodium acetate solution buffered to a pH of 4.5. They were vacuum embedded in 56° C. paraffin, sectioned at 5 to 8 microns and stained by the following methods:

a. Harris' Hematoxylin and Eosin.

b. Colloidal iron reaction.

c. Periodic acid-Schiff reaction.

d. Alcian blue 8GS reaction.

The colloidal iron procedure was that suggested by Rinehart and Abul-Haj.[2] The method of McManus[3] was used for the periodic acid-Schiff reaction. For each of these methods some sections were pretreated with testicular hyaluronidase and beta-glucuronidase, and some were subjected to peracetic acid oxidation as suggested by Fullmer,[4] followed or not by digestion with the aforementioned enzymes. Some of the sections for PAS staining were pretreated with malt diastase.

The alcian blue procedure was conducted under the principles established by Steedman (Pearse[5]) and Mowry.[6] Some

Fig. 1. A horizontal section of 8 microns through the footplate of a stapes showing replacement of practically all normal structure by otosclerotic bone. The section was stained by the colloidal iron method, and, although not apparent in the photomicrograph, there are areas of the bone matrix that stain more deeply than others. This indicates that intercellular substance varies in density in different parts of the otosclerotic lesion. (x 25)

Fig. 2. A horizontal vertical section of 8 microns in thickness through a footplate showing a marked positive reaction with colloidal iron procedure in otosclerotic areas. The larger area is anterior (A) while the smaller is posterior (P). There was a communication between the two in another section. A special filter was used to enhance the contrast of the reaction. (x 25)

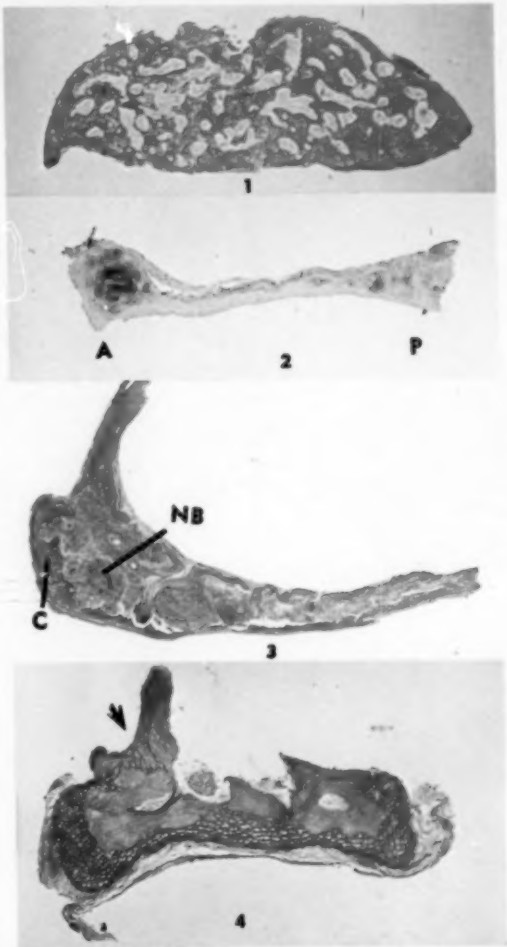


Fig. 3. A section through portions of the anterior crus and footplate showing marked proliferation of new bone (N.B.). It has invaded the cartilage (C.) and even replaced it in some areas. Numerous apposition lines are present with the colloidal iron stain followed by the PAS reaction. (x 25)

Fig. 4. A section through the footplate and a portion of the anterior crus. Cartilage (indicated by an arrow) near the base of the crus has become surrounded by new bone. Elsewhere in the footplate there is considerable irregularity in the bone. (x 25)

sections stained by this method were likewise pretreated with testicular hyaluronidase and beta-glucuronidase.

Undecalcified controls were not used for this series, because it was found by previous study[7] that the changes introduced by decalcification, at least as far as these particular procedures are concerned, were quantitative rather than qualitative and could, therefore, be considered as minor.

2. Fluorescence Microscopy. Eight specimens were fixed in absolute acetone immediately upon removal at the time of the operation. Undecalcified sections of 50 microns in thickness were obtained of each specimen by the procedure described by Frost.[8] This consists in grinding the bony fragments between two sheets of carborundum abrasive paper, grit 400. The specimens were kept wet with water during the entire procedure. They were washed in dilute detergent solution to remove adherent debris and then stained with acridine orange, rhodamine B, auramin O and fluorescein, respectively. The acridine orange method was that reported by von Bertalanffy and Bickis[9] and Armstrong.[10] The methods for the other fluorochromes were selected from experiments conducted by Vassar and Culling.[11] Rhodamine B was used in 1 per cent aqueous solution for sections pretreated with 1 per cent periodic acid for three minutes. Auramin O was also used in a 1 per cent aqueous solution without any accompanying oxidation. A 1 per cent solution of fluorescein in absolute alcohol was employed. After treatment with the respective fluorochrome the sections were examined with a Fluorestar microscope, the light source being an Osram HBO-200 mercury bulb.

3. Succinic dehydrogenase activity. Immediately upon removal, 12 specimens were placed into a vessel filled with liquid dichlorodifluoromethane (Freon 12). They were taken to the laboratory and remained for 120 minutes at 37° C. in a substrate solution containing buffered sodium succinate and nitro-blue-tetrazolium, prepared according to Nachlas *et al.*[12] An undecalcified section 50 microns in thickness was prepared of each specimen by Frost's method. Drops of the substrate solution were used instead of water. Following

this the sections were reincubated in the substrate for 30 minutes, air-dried and mounted in polyvinyl pirrolidone.

4. Cytochrome oxidase activity. Twelve specimens were stained for cytochrome oxidase by a method elaborated by Burstone.[13,14] Immediately upon removal they were placed in liquid dichlorodifluoromethane for about one hour and were then incubated for 120 minutes in a solution containing a suitable substrate and N-phenyl-p-phenylenediamine. 1-hydroxy-2-naphthoic acid was used as a substrate for the first four experiments, 8-amino-1,2,3,4-tetrahydroquinoline was used as substrate for the remaining eight specimens. Sections of about 50 microns in thickness were then prepared by Frost's technique, with the use of the substrate solution in place of water, and reincubated for 30 minutes. They were transferred to a buffered cobaltous acetate-formalin solution for one hour and were then air-dried and mounted in polyvinyl pirrolidone. A small amount of cytochrome C was added to the substrate to enhance the reaction for 10 of the specimens.

5. Alkaline phosphatase activity. Twelve specimens, immediately after removal at the time of surgery, were placed in absolute acetone at 4° C. and subsequently prepared by Frost's method to yield sections of about 50 microns in thickness. Cold acetone was used as the cooling medium while grinding. The sections were incubated overnight in a substrate solution containing naphthol AS-MX phosphate, dimethylformamide, tris (hydroxymethyl) aminomethane buffer of pH 8.3 and, for eight of the specimens, fast violet B diazonium salt, according to the principles established by Burstone.[15] For the other four specimens the post-coupling technique was used. The sections were placed in an adequate diazonium salt solution after coupling in a substrate devoid of this salt. Either fast red TR or fast violet B diazonium salt was used for the post-coupling procedure. Polyvinyl pirrolidone was used as mounting medium for all sections.

6. Acid phosphatase activity. Twelve specimens were fixed in absolute acetone at 4° C. and similarly prepared by Frost's procedure. They were incubated in a substrate solution[16]

similar to the one used for alkaline phosphatase but in which an acetate buffer of pH 5.4 was substituted for the tris buffer. Manganese chloride was added to the solution as an activator. All sections were processed by simultaneous coupling with fast violet B diazonium salt. After incubation the sections were air-dried and mounted in polyvinyl pirrolidone.

7. Controls. For each of the succinic dehydrogenase, cytochrome oxidase, alkaline and acid phosphatase methods, one specimen was placed in boiling water for three minutes prior to its incubation in the respective substrate solution. From this point they were prepared in a manner identical to that described above. For the succinic dehydrogenase method one additional specimen was specifically inhibited with monoethyl oxalacetic acid.

Four specimens were fixed in 80 per cent alcohol at 4° C. for 24 hours and decalcified by chelation with ethylenediamine tetracetic acid. This method, described by Freiman,[17] is supposed to be suitable for enzymatic studies. The specimens were reactivated in a sodium barbiturate solution, vacuum-embedded in 56° C. paraffin, sectioned at 5 microns and then incubated in the specific substrate solution for each of the four enzymatic tests performed.

Two specimens were fixed in neutral 10 per cent formalin for 24 hours at 4° C. and decalcified in a buffered 5 per cent formic acid solution, as described by Schajowicz and Cabrini,[18] until all calcium had been eliminated. They were vacuum-embedded in paraffin and the blocks sectioned at 5 microns. The sections were stained for acid phosphatase by the procedure previously described.

Finally two specimens were placed in liquid dichlorodifluoromethane immediately after removal and were prepared by freeze-drying. The frozen-dried specimens were vacuum-embedded in paraffin and sectioned by the Adamstone-Taylor cold-knife technique at 8 microns. The sections were activated with sodium barbiturate and submitted to procedures similar to those described for the specimens prepared by Frost's method.

FINDINGS.

Sections of all 45 specimens in the first series were stained according to each of the methods previously mentioned and a comparison of observations made.

The heads and posterior crura were found to be devoid of otosclerotic involvement in all specimens. The anterior crura showed otosclerotic lesions in approximately one-half (23) of the series. Changes in vascular distribution and in the interglobular spaces could be seen in all specimens. These commonly extended throughout the whole of the ossicle not involved with typical otosclerotic lesions.

The footplates showed variable degrees of involvement. Six were extensively involved. There were large and numerous connective tissue spaces and increased cellularity (see Fig. 1). Twenty-one were involved with less marked changes. The amount of cellularity was less pronounced, and the connective tissue spaces were smaller and less numerous. The remaining 18 specimens showed localized otosclerotic foci. These were anterior in 15, peripheral in two and posterior in one footplate. In a few specimens with anterior foci only the superior (facial) half was involved.

The non-involved areas of these footplates revealed the same changes in vascular distribution. The distribution of the constituent elements was distorted except in areas adjacent to blood vessels and connective tissue spaces, where a normal lamellar structure was apparent. Large areas of normal bone with normal stapes architecture and blood vessel distribution were found in only three footplates. In 35 footplates cartilage was present in portions of the ligament and vestibular margin.

1. Mucopolysaccharides. The bone matrix of the footplates was found to be PAS positive in all specimens. Twenty-one specimens revealed areas of stronger reaction against a less reactive background. These were usually found about the connective tissue spaces and along the cement lines. The reaction became somewhat less intense in sections pretreated with hyaluronidase and beta-glucuronidase. In the sections

oxidized with peracetic acid the effect of the enzymes was negligible.

There was good correlation between the results with the colloidal iron and alcian blue procedures. A positive reaction was obtained in only 22 specimens and in most cases consisted of dispersed granules disseminated throughout the bone matrix. In four specimens, however, there was a significant amount of positive reaction in well limited areas with severe otosclerotic involvement (see Fig. 2). When a PAS counterstain followed the colloidal iron procedure it was seen that the lines of apposition were PAS positive but these were not present with colloidal iron or alcian blue procedures (see Fig. 3).

The cartilaginous portions of the footplates were consistently PAS positive and in all but six specimens yielded positive reactions with the colloidal iron and alcian blue. The degrees of reaction varied with the latter procedure. Very strong positive reactions with colloidal iron were seen in specimens for which the cartilage was in active proliferation. Irregularity in size and distribution of its cells, distorted nuclei and increased cellularity of the cartilage were present in these specimens. In two footplates growth of otosclerotic bone around the cartilage was observed (see Fig. 4); in one specimen, invasion of the footplate cartilage by otosclerotic bone from the facial margin of the oval window was seen. The results in stainability with the alcian blue procedure were not so striking as those with colloidal iron. In the specimens for which the cartilage showed no reaction with either colloidal iron or alcian blue, calcification was present. This was judged by the hematoxylin and eosin preparations.

The sections subjected to hyaluronidase digestion followed by the colloidal iron method exhibited little or no reaction. Glucuronidase, as expected, was less effective in reducing the reaction; nevertheless, a significant decrease could be noticed. The peracetic acid oxidation, on the other hand, contributed some enhancement. Digestion of the preoxidized sections proved to be practically ineffective, particularly when glucuronidase was used. Both enzymes were effective in reduc-

ing the intensity of the alcian blue reaction of which hyaluronidase was the most effective.

The osteocytes showed the characteristic changes in shape, distribution and relative number usually found in otosclerosis. Their cytoplasm was usually PAS positive. This was not glycogen because it persisted in the specimens pretreated with malt diastase.

The connective tissue spaces revealed variable degrees of activity. Osteoclasts were occasionally seen in the bony interfaces of the heavily involved otosclerotic footplates. Fibers and a small amount of PAS positive intercellular substance surrounded the connective tissue cells which seemed to be more numerous in the specimens with severe involvement. The intercellular substance exhibited a slightly positive reaction with both colloidal iron and alcian blue procedures in most preparations.

2. Fluorescence Microscopy. Sections of otosclerotic bone obtained by grinding were of the order of 50 microns in thickness. Although other bony specimens could be thinned to as little as 3 microns by this procedure, it was found that the footplates in particular disintegrated completely with attempts to make them thinner. In spite of the thickness these sections revealed adequate histological detail with cells and canaliculi well individualized.

The specimens stained with acridine orange were viewed with the use of a Corning exciter filter of 5-58 color specification and a Kodak Wrattan G yellow barrier filter. Green fluorescence of the bone matrix was obtained by this procedure while blood vessels and connective tissue spaces remained dark. The fluorescence of the areas heavily involved by otosclerosis was much stronger than that of other areas of the bone (see Fig. 5). Red fluorescence would indicate concentrations of ribonucleic acid, but it was not present.

The specimens prepared by the periodic acid-rhodamine B procedure were viewed with the use of a Corning exciter filter peaking at 365 mu. and a white barrier filter. Reticular fibers yielded an orange fluorescence, while collagen was in

green. The specificity of this method is not known. In the present series the matrix of the bone fluoresced in purple, and a green fibrillar network was seen that exhibited light fluorescence. The cells were dark (nonfluorescent), and the connective tissue spaces showed green and orange fibrils and dark cells. In lightly involved footplates both bone and connective tissue cells showed red nuclei and dark, nonfluorescent cytoplasm.

Fig. 5. A ground section of a footplate prepared by the method of Frost and stained with acridine orange. The area involved with otosclerosis fluoresces intensely in green. The remainder of the footplate does not show the same amount of fluorescence. There was no red fluorescence to indicate presence of RNA. (x 68)

The specimens stained with auramin O were observed with the use of a 5-58 exciter filter and a clear barrier filter. This procedure revealed collagen fluorescence in green, and elastic fibers and muscle in yellow. Pretreatment of the specimens with 5 per cent ferric ammonium sulfate inhibited the fluorescence of collagen fibers, while that of the elastic remained. In this series both cartilage and otosclerotic bone showed dispersed light green fluorescence while non-otosclerotic bone did not fluoresce. In the mordanted slides all fluorescence disappeared.

For observation with fluorescein the 5-58 exciter and clear barrier filters were also employed. This method showed reticular fibers that fluoresced in yellow. The specificity of the reaction is not known. Thin yellow fibers were observed in the matrix of the cartilage and the intercellular substance of the connective tissue spaces. Some areas of the otosclerotic bone exhibited mild yellow fluorescence. Figure 6 shows bone with a variable amount of fluorescence between uniformly

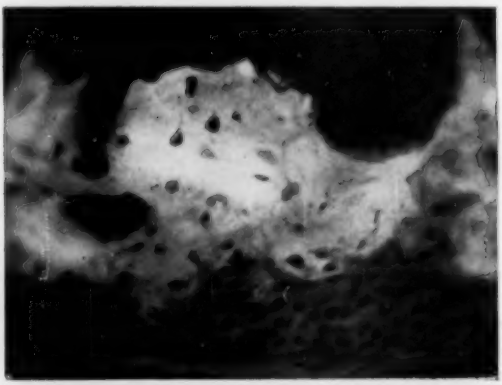


Fig. 6. A ground section of a part of the footplate prepared by the method of Frost and stained with fluorescein. The otosclerotic bone fluoresces in yellow in variable amounts. The cartilage fluoresces uniformly and connective tissue spaces show fluorescent fibers. (x 360)

fluorescent cartilage and dark connective tissue spaces containing fluorescent fibers.

3. Succinic Dehydrogenase. A positive reaction for succinic dehydrogenase was obtained for the mucosa, blood vessels and connective tissue spaces in all otosclerotic specimens. The same positive reaction was found to occur in the head and crura (see Fig. 7). In the footplates, intensely stained positive osteoclasts were occasionally seen; in heavily involved footplates, the osteocytes in close proximity to blood vessels, were mildly positive. The reaction was not so intense but was qualitatively identical in the EDTA decalcified sections. The frozen-dried sections were morphologically inferior to

the fresh ground preparations but exhibited a comparable amount of positive reaction. No reaction was obtained in the specimen that was boiled or in the one that had been specifically inhibited with monoethyloxalacetic acid.

4. Cytochrome Oxidase. Some positive reaction was obtained for the middle ear mucosa and the lining of the small blood vessels. Osteocytes in the heads and crura did not show any reaction except for one specimen in which the anterior crus was involved with the disease (see Fig. 9). In

Fig. 7. A ground section of head, crura and part of the footplate (upper right) prepared for the demonstration of succinic dehydrogenase. The reaction is essentially limited to mucosa, blood vessels, fibrous connective tissue and osteoclasts. The blackened area in the footplate is not a reaction but "air spaces" due to the grinding. The vascular channels in the footplate were numerous and gave a markedly positive reaction. (x 25)

the footplates many cells exhibited positive reaction in variable degrees (see Fig. 8). Osteoclasts showed the strongest degree of reaction. Fibroblasts of the connective tissue spaces reacted mildly. Typical osteoblasts were not seen. The reaction was qualitatively equivalent for each of two substrates but was more intense for the specimens coupled with 8-amino-1,2,3,4-tetrahydroquinoline. There was little quantitative difference between these specimens and the frozen-dried prep-

arations. The reaction was poor in the EDTA sections and totally negative for the boiled control.

5. Alkaline Phosphatase. The middle ear mucosa was strongly positive with the alkaline phosphatase procedure. Blood vessels were equally positive. The matrix of the bone did not stain. Osteocytes of the head and crura were essentially negative. In the footplate a few cells, particularly those in the proximity of blood vessels, revealed strong posi-

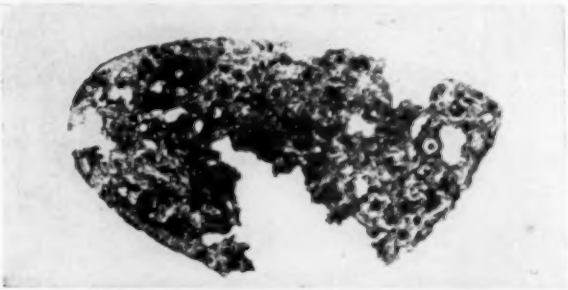


Fig. 8. A horizontal ground section of a stapes footplate prepared for demonstration of cytochrome oxidase activity. The dark areas are those of very positive reaction. Although most of the footplate was comprised of otosclerotic bone, the more central portion produced a more marked reaction. The reaction occurred in the mucosa, blood vessels, fibroblasts and osteoclasts with some degree of reaction in osteocytes of the otosclerotic focus. (x 25)

tive reactions. Other osteocytes produced a mild reaction. In heavily involved otosclerotic areas of footplates no positive reactions were seen, although some were usually present in the surrounding cells. Blood vessels were more numerous and more intensely positive in these footplates. Osteoclasts did not react, and osteoblasts were not seen. The reaction was slightly more intense for the simultaneous coupling procedure than for the post-coupling. There was little difference between these results and those of the frozen-dried specimens except that a precise localization of particular areas was more difficult. The specimens decalcified by chelation in EDTA yielded less reaction but were qualitatively similar. No reaction was observed in the boiled control.

6. Acid Phosphatase. A mild reaction was observed for the middle ear mucosa. The osteocytes of the heads and crura showed no reaction. Those of the footplates occasionally revealed some mild activity but were mostly negative. Osteoclasts showed an intense positive reaction. Some blood vessels revealed mild activity. The bone matrix in the areas markedly involved with otosclerosis exhibited a fair amount of reaction. This was not apparent in other areas of the foot-

Fig. 9. A ground section of a stapes prepared for demonstration of cytochrome oxidase activity. The base of the anterior crus shows a marked positive reaction in the area comprised of otosclerotic bone. There is also a positive reaction in the contents of the connective tissue spaces elsewhere and in the base of the posterior crus. (x 25)

plates or in the heads or crura. The reaction was essentially identical in the ground and frozen-dried material. The specimens decalcified by chelation were not adequate. Those totally decalcified in buffered formic acid were qualitatively similar to the non-decalcified specimens, but the reactions were much less intense and often difficult to interpret. No reaction was obtained from the boiled controls.

DISCUSSION.

The bone of the otic capsule and ossicles is in many as-

pects different from other bones in the organism. The hypothesis[19,20,21] has been frequently suggested that the enchondral bone of the capsule represents an arrested stage in development of bone in cartilage. This has been extended to structures closely related that are not a part of the capsule, such as the external bone of the stapes footplate and the head and crura of the stapes. These structures of branchial origin reveal evidence for developmental arrest, such as the absence of Haversian architecture. Otosclerosis is a disease of the bone of the otic capsule. These facts should be kept in mind, for the interpretation of findings must rely upon findings for normal bone in areas other than the otic capsule. The unavailability of normal specimens (control) that could be prepared under identical conditions renders the interpretation of findings for this series more difficult, since little is known about bone of the otic capsule from a histochemical point of view. The crura and the areas of the footplate not involved by otosclerosis are not ideal controls, since changes in cellularity and vascularity of the type described by Covell and Feinmesser[22] for the malleus and incus were invariably present.

A unique characteristic of bone as compared to other animal tissues is its hardness because of the deposition of chemically complex minerals in an equally complex organic matrix. The hardness of bone renders ordinary histological methods unsuitable for its study. Decalcification, or rather demineralization, is necessary before the conventional histological methods can be used.

For many histochemical methods decalcification has to be avoided and, in other instances, there is some controversy concerning its effects. The localization of acid phosphatase, for example, by some authors[18] is best determined after complete decalcification, until no trace of calcium can be found in the bone. Others[16] hesitate to use decalcification. For some methods, the use of chelating agents is feasible, but for others it is not satisfactory.

Preparation of the specimens by freeze-drying and sectioning by the cold-knife technique was the first method contem-

plated for these studies. Better over-all morphology, however, was achieved in subsequent experiments with finely ground specimens, although precise enzyme localizations were somewhat clearer in the frozen-dried material. Both of these techniques permit an opportunity for study of mineral and organic constituents of bone.

Most of the organic matrix of bone is comprised of collagen probably secreted by the osteoblasts. The remainder is intercellular or ground substance which is roughly about 5 per cent of the organic matrix. As in all connective tissues, the intercellular substance of the bone presents itself in various degrees of polymerization, ranging from the consistency of interstitial fluid to that of condensed basement membranes. Through its interconnection with tissue fluid the intercellular substance is intimately associated with ion exchanges and nutrition of bone.[23] Other constituents of the intercellular substance include enzymes, other proteins and polysaccharides.

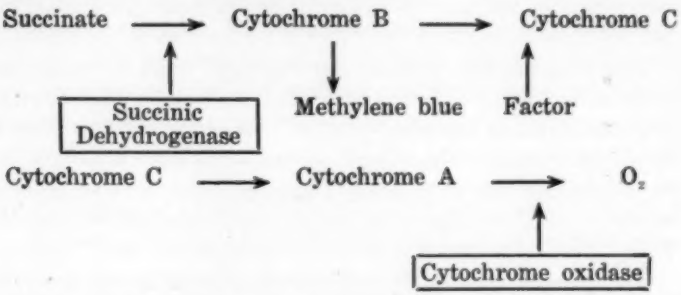
The present day interest has centered upon a group of substances, the acid mucopolysaccharides, which play a very important role in the physiology of connective tissues. From the histochemical point of view there is still some controversy on the methods utilized to demonstrate these complex carbohydrates. The alcian blue reaction seems to be regarded by most authors as the most specific of the available tests.[6,24]

An attempt was made to determine whether changes in the degree of polymerization of the intercellular substance could be observed in the otosclerotic bone. This was not present in the head and crura of the stapes. It was observed in well defined areas of four of the six highly involved footplates. In 18 other footplates there were only dispersed granules that showed positive reactions after alcian blue and colloidal iron procedures. It then seems probable that in extensive otosclerotic lesions the intercellular substance tends to be in a more condensed state and probably is less dynamic. The cartilage of the footplate, on the other hand, seems to show an increase in reactions in early stages, and a loss of the positive reaction occurs later in the disease. Markedly involved otosclerotic footplates are often devoid of cartilage.

These changes in the cartilage are in good agreement with the observations of Schajowicz and Cabrini[25] who reported an increase of thionine metachromasia in proliferating cartilage that disappeared after calcification of the cartilage.

The fluorescence technique was attempted mainly to evaluate whether larger concentrations of cytoplasmic RNA would be apparent in the bone cells. This was found not to be the case. It was observed, however, that the otosclerotic bone had more affinity for the fluorescent dyes than bone with little or no pathological involvement. This may be related to the state of aggregation of the intercellular substance.

One of the important enzyme systems involved in cell nutrition is the succinic oxidase system that comprises the main components of normal oxidation.[5] The system may be represented schematically as follows:



Studies of this system in normal bone[26,27] have shown that osteoclasts exhibit intense succinic dehydrogenase and cytochrome oxidase activity, while osteoblasts show little if any succinic dehydrogenase and mild cytochrome oxidase activity. Intense succinic dehydrogenase activity of osteoclasts was also described for giant-cell tumors of bone (osteoclastoma).[24]

In the present series the osteoclasts showed comparable respiratory enzyme activity. In addition, some osteocytes in close proximity to blood vessels exhibited mild succinic dehydrogenase activity. Since typical osteoblasts were not seen in these specimens it seems possible that these cells may be engaged in blastic activity. These findings suggest that sites

of high metabolic activity are present in areas closely related to the otosclerotic foci. It is not clear whether this is directly due to the disease process or an attempt to remove diseased bone.

Mineral deposition on the organic matrix of bone, like nutrition and respiratory activity, is also to be considered as dynamic metabolic activity. The phosphatases are enzymes concerned with phosphate transfer and, therefore, with this activity. Although a number of specific phosphatases exist they are commonly studied in two groups, alkaline and acid phosphatases. The greater amount of histochemical work has been concerned with these groups, although tests for a few specific phosphatases are available.

In normal bone it has been shown[17,25] that alkaline phosphatase activity is predominant in cartilaginous calcification, formation of the bone matrix and calcification of the matrix. Osteoblasts consistently exhibit a high degree of activity. Acid phosphatase activity, on the other hand, has been demonstrated for the osteoclasts and areas of bone resorption.[16,18]

In these experiments little or no alkaline phosphatase activity was present in the areas highly involved with otosclerosis. Around these areas, however, osteocytes showed variable degrees of activity. Typical osteoblasts were not seen. It is interesting to note that in other studies on bone of different pathological conditions[25] the observation was made that all alkaline phosphatase activity was lost when osteoblasts were present in the osteoid intercellular substance. The ability of the footplate osteocytes to return to a metabolically active condition is possibly a consequence of their embryonic origin and arrested development. If it may be safely assumed that this enzyme activity implies new bone formation, it is significant that the reaction occurred only in those specimens exhibiting extensive otosclerotic involvement and increased vascularity. Cells of the heads and crura and non-involved areas of the footplates did not react.

Acid phosphatase activity in these specimens was present in the matrix of the otosclerotic bone as well as in the osteo-

clasts. The significance of the former finding is difficult to evaluate.

It is apparent then that the otosclerotic bone is metabolically active particularly during the stage of growth and invasion. Bone formation and resorption seem to be invariably present except in the highly involved areas where activity ceases. When metabolic activity is present, as judged by these enzymatic processes, it resembles that of normal growing bone. No gross physiological disturbances could be demonstrated by these techniques.

SUMMARY.

One hundred and ten stapes were made available for study by the stapes replacement operation. They were subjected to various histochemical and other technical procedures in an attempt to study further the nature of otosclerotic bone.

Forty-five of the specimens were fixed, decalcified, embedded in paraffin, sectioned at 5 to 8 microns and stained by routine and special methods. The head and posterior crus were devoid of otosclerotic foci in all specimens. Twenty-three showed foci in the base of the anterior crus. All of the footplates revealed otosclerotic bone. In six, the lesions were extensive; in 18, there were localized areas of foci, and in 21, there were less marked pathological changes. Thirty-five footplates revealed the presence of cartilage in portions of the vestibular margin and ligament. Changes in vascularity and interglobular spaces were present throughout most specimens.

The bone matrix of the footplates was PAS positive. The reaction was somewhat diminished after pretreatment with hyaluronidase and beta-glucuronidase. The colloidal iron and alcian blue procedures were positive in the form of granules dispersed throughout the bone matrix of 22 specimens. In four, the positive reaction was chiefly in the areas of severe otosclerotic involvement. Hyaluronidase was more effective in reducing the intensity of the alcian blue reaction than was beta-glucuronidase. The cytoplasm of osteocytes of the otoscle-

rotic bone was usually PAS positive. The staining persisted after pretreatment with malt diastase. A small amount of PAS positive intercellular substance was present about the connective tissue cells of the lesion; it was also slightly positive with colloidal iron and alcian blue.

Ground sections of eight undecalcified stapes were studied by means of fluorescence microscopy. Acridine orange produced a green fluorescence of bone matrix with a much stronger fluorescence of otosclerotic bone. Periodic acid-rhodamine B produced an orange fluorescence for reticular fibers, green for collagen and purple for bone matrix. Auramin O produced a green fluorescence for collagen and yellow for elastic fibers. Cartilage and otosclerotic bone fluoresced in a light green. This was not apparent in non-otosclerotic bone. Fluorescein yielded a yellow fluorescence for reticular fibers, and some areas of otosclerotic bone showed a less intense yellow fluorescence.

Succinic dehydrogenase was intensely positive for blood vessels and connective tissue spaces of otosclerotic foci. Osteoclasts were positive, and osteocytes in the vicinity of blood vessels were mildly positive.

Cytochrome oxidase was present in middle ear mucosa and the lining of small blood vessels. Osteoclasts showed the strongest reaction, but there were varying degrees of reactions in other cells of the otosclerotic lesion.

Alkaline phosphatase was strongly positive in the middle ear mucosa and about blood vessels. Osteocytes in the vicinity of blood vessels were slightly positive.

Acid phosphatase reaction was mild in the middle ear mucosa and for some blood vessels. An intense positive reaction was present in osteoclasts with a fair amount in the matrix of the otosclerotic bone.

The authors are indebted to Doctor Theo. E. Walsh for the specimens of stapes removed at the time of the replacement operation and are grateful for the technical assistance of Kathleen Tinglin in carrying out many of the tests.

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ENDOSCOPIC DIAGNOSIS AND STEROID AND ANTIBIOTIC THERAPY OF ACUTE LYE BURNS OF THE ESOPHAGUS.*

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The ingestion of any alkali frequently results in burns of the mouth, pharynx and esophagus. Extensive burns of these areas usually have a poor prognosis since severe corrosive esophagitis results in stricture formation; however, the absence of oral lesions does not indicate that the esophagus has been spared. It is, therefore, important to determine the extent and severity of esophageal involvement since prophylactic methods can be used to prevent stricture formation.

Prophylactic bouginage was the method most frequently advocated from 1925 to 1950. During the last decade, the use of steroids and antibiotics as a prophylaxis has gained in popularity. In assessing the prophylactic value of either of these methods, many previous reports assumed that all patients who ingested alkali had sustained esophageal burns. This report will discuss the use of esophagoscopy in the diagnosis of esophageal burns and, in light of the endoscopic findings, evaluate the administration of steroids and antibiotics in the management of 53 patients.

METHOD OF MANAGEMENT.

All patients in this series were treated with steroids and

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antibiotics, immediately upon admittance to the hospital and until the extent and severity of the esophageal damage could be determined. They received 100 - 150 mg. of Cortisone a day in two or three divided doses. Hydrocortisone and prednisone in equivalent dosage were sometimes used in place of Cortisone with similar results. The patients with severe oral burns received parenteral steroids for 48 - 72 hours, after which time the oral edema and sialorrhea usually diminished sufficiently to allow the patient to take fluids as well as steroids by mouth.

Similarly, oral or parenteral antibiotics were given in conjunction with the steroids. Penicillin, streptomycin and chloramphenicol were the parenteral antibiotics most frequently used. Penicillin was usually administered in a dosage of 800,000 to 1,200,000 units daily. Streptomycin and chloramphenicol were given in 25 - 50 mg./kg. body weight/24 hours in divided doses. In most cases, at least two of the antibiotics were used in combination. Tetracycline or chloramphenicol, given in 25 - 50 mg./kg. body weight/24 hours in divided doses, were the two antibiotics most frequently administered orally.

During the first two years of this study, all patients received steroids and antibiotics for two weeks and then an esophagram with barium was made followed by an esophagoscopy. Since less than 50 per cent of the patients sustained esophageal burns, the disadvantages of this method became obvious.

In order to evaluate the efficacy of steroids and antibiotics, the following method of evaluating the extent and severity of the oral and esophageal burns was established. The lips and oral cavity of all patients were examined for hyperemia, edema and ulceration of the mucosa as soon as they were seen by a physician. A Beck pharyngoscope was used to evaluate the larynx and hypopharynx. When the hypopharynx appeared free of burns, or first and second degree oral burns were seen, a careful esophagoscopy was done within 24 - 48 hours after the ingestion of the caustic. The esophagoscope was passed cautiously until an area of

burn or ulceration was seen and then withdrawn. Patients with esophageal burns were continued on steroids and antibiotics for two weeks. The patient was then re-evaluated by esophagram and esophagoscopy. If the esophagus was free of any burns, steroids and antibiotics were discontinued, and the patient was discharged.

When third degree burns of the hypopharynx were present and especially with burns involving the larynx, no esophagoscopy was carried out, but the patient was continued on steroids and antibiotics until his condition had improved clinically, which was usually 14 - 21 days after the ingestion of the alkali. Patients with these severe hypopharyngeal and laryngeal burns had roentgenological examination of the esophagus, first without, and then with barium contrast in the esophagus, prior to esophagoscopy.

RESULTS OF MANAGEMENT.

All 53 children had a history of ingestion of caustic. Twenty-eight of these children were admitted to Homer G. Phillips Hospital between October, 1953, and June, 1956, and 25 were admitted to St. Louis Children's Hospital during a seven-year period beginning in January, 1954. In all of these cases the caustic ingested was lye (NaOH). The lye was in a crystal or powdered form in 48 instances and in a liquid form when ingested by five patients.

Physical examination of all the children revealed hyperemia and/or edema, sialorrhea, ulceration or bleeding of the oral cavity. Twenty-nine of these patients were males and 24 females. Sixteen of the children were Caucasian and 37 were Negro. Their ages ranged from 10 months to four years with an average age of 25 months. One mentally retarded patient was six years of age when he ingested lye. Of the total of 53 patients treated with steroids and antibiotics, 47 were esophagoscoped, revealing burns in 21 (44.7 per cent). Table 1 summarizes the findings.

Hyperemia of the esophageal mucosa with superficial desquamation of the epithelium indicated a first degree burn.

Superficial blisters, ulcers, hyperemia and patchy membranous exudate of the mucosa were characteristic of second degree burns. Areas with deep loss of esophageal epithelium, which were hyperemic with evidence of granulation tissue, were classified as third degree burns. Esophagrams with barium were made on 39 of these 53 children. Narrowing of the esophagus, indicating early stricture formation, was seen on three esophagrams. This narrowing of the esophagus was not demonstrable until two to four weeks after the ingestion of caustic. Eighteen of the 21 patients with positive esophagoscopies were followed for 12 - 18 months and then

TABLE I.

Summary of Esophagoscopy Examination and Stricture Formation.

	*H.G.P.	†S.L.C.H.	Total	Strictures
Negative Esophagoscopies	15	11	26	0
Positive Esophagoscopies				
1° Burns	3	10	13	0
2° Burns	3	2	5	0
3° Burns	1	2	3	3
	7	14	21	3

*Homer G. Phillips Hospital.
†St. Louis Children's Hospital.

re-evaluated by esophagram as well as an esophagoscopy. Fifteen of the 18 patients had no evidence of stricture 12 - 18 months after their initial burn. Three patients developed strictures within four to eight weeks after ingestion of the caustic. Eight of the 26 patients with negative findings by esophagoscopy following ingestion of lye were re-examined by endoscopy 12 - 18 months later; all were found to be normal, and none developed strictures later.

The following is a brief summary of the clinical courses of each of the three patients who developed strictures:

Patient 1, J. M., a two-year-old Negro male, ingested lye in a powdered form. On admittance to the hospital, two hours later, the mouth revealed first and second degree burns of the anterior and posterior pharynx. He was placed on steroids and antibiotics. Roentgenograms taken six hours after admittance revealed air in the retropharyngeal space, indicating spontaneous perforation of the posterior pharynx. This air reabsorbed within 24 hours. The following day pharyngoscopy revealed first, second and third degree burns of the hypopharynx and larynx, including false and true vocal cords and aryepiglottic folds. A gastros-

tomy was performed 48 hours after admittance. Eight days after admittance the patient developed mediastinal emphysema, a left tension pneumothorax and subcutaneous emphysema. Exploration of the esophagus and cervical regions failed to reveal the site of perforation. The air gradually reabsorbed over the next ten days. Thirty-three days after the burn, an esophagram with barium was interpreted as normal. Forty-seven days post ingestion, esophagoscopy revealed the larynx to be normal; but the middle third of the esophagus was narrowed, and there was an area of granulation tissue seen at the level of the cricopharyngeus. Re-evaluation by esophagoscopy 68 days after the burn demonstrated further constriction of the middle third of the esophagus. Steroids were discontinued, and 75 days post ingestion the lower two-thirds of the esophagus was removed and esophagogastrostomy performed. Unfortunately, the site of anastomosis has required dilatation every three to four months during the past two and one-half years. Pathological examination of the excised esophagus revealed "extensive loss of the muscularis and replacement of lumenal epithelium with granulation tissue."

Patient 2, H. M., was a three-year-old Negro boy who ingested liquid lye. On admittance to the hospital three hours later, first, second and third degree burns of the mouth were seen. Steroids and antibiotics were begun immediately. Sixteen days post ingestion an esophagram with barium revealed narrowing of the middle third of the esophagus. Esophagoscopy on the seventeenth post ingestion day revealed burns of the epiglottis, pharynx and narrowing of the cricopharyngeus as well as the middle third of the esophagus. Repeat esophagrams and esophagoscopies continued to demonstrate further narrowing of the esophagus. Forty-six days post ingestion a colon transplant was performed with anastomosis of the transverse colon to the stomach, and the cecum to the pyriform sinus. Twenty-six days after the colon transplant, the epiglottis was removed because of pharyngeal obstruction. Details of this surgical procedure have been previously reported.[1] This patient has continued to eat very well without requiring dilatation, for the past two years.

Patient 3, K. H., was a three and three-fourths-year-old Negro boy who ingested lye in a liquid form. On admittance to the hospital three hours after ingestion, examination revealed first and second degree burns of the pharynx, hypopharynx and oral cavity. Steroids and antibiotics were begun, and on the eleventh post ingestion day an esophagram with barium was interpreted as normal. Consent for an esophagoscopy could not be obtained. Steroids and antibiotics were discontinued on the fourteenth post ingestion day. Twenty-nine days after ingestion, he developed dysphagia, and the following day an esophagram revealed a stricture of the hypopharynx. A gastrostomy was performed 35 days after ingestion of the alkali. Despite many attempts nothing could be threaded through the esophagus from either the distal or proximal end. He continued with a gastrostomy for five years. During this time he became a difficult behavioral and nutritional problem. One year ago a colon transplant was done followed three weeks later by a pharyngoplasty. A sheet of dense fibrous tissue partially obliterating the larynx was removed with the epiglottis and a skin graft placed in the pharynx. The patient had no evidence of stenosis at the site of anastomosis one year later. The psychological and nutritional improvement in this patient has been remarkable.

DISCUSSION.

Krey[2] states that the severity of esophageal injury is de-

pendent upon the concentration of the alkali ingested. Others feel that the type and amount of alkali ingested are also factors in determining the extent of esophageal damage. Since it is rare that parents can provide this information, other methods must be used to assess the esophageal damage. In our experience, a careful esophagoscopy with direct visualization of the esophagus is superior to any other method presently available.

Early esophagoscopy has two advantages: *1.* The patients without esophageal burns which comprised 55 per cent of our series can avoid prolonged hospitalization. *2.* Esophagoscopy provides some knowledge of the extent and severity of the esophageal damage. The limitations of esophagoscopy must also be recognized and accepted and appear to be the following: *1.* It is truly difficult to evaluate the depth of any burn with absolute certainty by observing superficial epithelial necrosis. *2.* When a severe burn is encountered in the upper third of the esophagus, the esophagoscope is not passed beyond this area, and the involvement of the middle and lower thirds is not known. *3.* Occasionally, and when directly visualized early, the esophagus may be interpreted as normal with, nevertheless, later development of stricture, as was reported by Cleveland.[3] Esophagrams may be helpful in evaluating stricture formation, but in our cases esophagoscopy revealed developing strictures while esophagrams made concurrently were interpreted as normal.

Until recently, prophylactic bouginage as advocated by Salzer[4] and Bokay[5] was used by many pediatric centers[6,7,8] to treat children who had ingested lye. This method was considered effective in the prevention of marked strictures, but the prospect of repeated dilatations for several years in some patients was never eagerly anticipated by patient or physician.

Following reports by Spain[9,10] that the early administration of Cortisone had an anti-inflammatory effect and inhibited fibroplasia in wounds, Rosenberg[11] used Cortisone to attempt to prevent stricture formation in rabbits with experimental lye burns. His rabbits succumbed to severe suppurative com-

plications, so Rosenberg[12] then gave the rabbits antibiotics in conjunction with steroids and was successful in reducing the incidence of esophageal stenosis in these animals. Similar findings were reported by Floberg and Koch[13] using cats, and by Weisskopf[14] using dogs. Shortly thereafter there were several reports of successful treatment by this method.[14,15,16] Subsequently, Ray and Morgan[17] used steroids and antibiotics to treat 11 patients, with one developing stricture. Cleveland[3] treated 23 patients, with five developing strictures; Yurich[18] treated ten patients with caustic esophageal burns, and only two developed strictures.

Most reports stress the importance of steroids in reducing the incidence of esophageal stenosis, but the need for intensive antibiotic therapy is emphasized by Bosher[19] in discussing the significant role that bacterial invasion of the esophageal wall may play in the pathogenesis of the lye burn and stricture formation.

Previous reports concerning the incidence of esophageal stenosis in children and those assessing the effectiveness of prophylactic methods used to prevent strictures, must be re-evaluated in view of the recent esophagoscopy findings of Alfred and Harris[20] who reported that 33 per cent of their patients with oral burns had esophageal burns. Yurich[18] found esophageal burns in only ten of 37 patients with oral burns. Similarly, 21 of 47 patients esophagoscoped in our series were found to have esophageal burns. It is apparent that no longer can one assume that all patients with oral burns sustain esophageal burns.

In our series, steroid and antibiotic therapy was found most useful in patients with first and second degree esophageal burns. This therapy also eliminated the necessity of early repeated bouginage, as had previously been advocated at our institution,[8] in 18 patients with first and second degree burns. Experimental[19] and clinical evaluation[21] has indicated that severe burns which involve the muscularis are usually irreversible, and restoration of normal deglutition is rare without repeated dilatations or surgical removal of the stricture.

SUMMARY.

1. Fifty-three patients with oral burns following ingestion of lye were evaluated by esophagoscopy or esophagram. Of 47 patients esophagoscoped only 21 (44.7 per cent) were found to have esophageal involvement.

2. All 21 patients were treated with steroids and antibiotics. A follow-up 12 - 18 months later revealed that only three patients with third degree esophageal burns had developed strictures, all within two months following ingestion.

3. In first and second degree burns of the esophagus, stricture formation was prevented by using antibiotic and steroid therapy only.

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NEW YORK EYE AND EAR INFIRMARY ALUMNI MEETING.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 9-11, 1962.

Symposia will be offered on "Complications of Stapes Surgery" and "Significance of a Mass in the Neck." There will also be a Closed Circuit Television Demonstration of surgical procedures and lectures.

Additional information may be obtained by writing to Dr. Merrill Goodman, Secretary, Alumni Association, 218 Second Avenue, New York 3, N. Y.

SURGICAL EXPOSURE OF THE INTERNAL AUDITORY CANAL AND ITS CONTENTS THROUGH THE MIDDLE, CRANIAL FOSSA.*

WILLIAM F. HOUSE, M.D.,

Los Angeles, Calif.

The VII and VIII cranial nerves and the internal auditory canal are traditionally surgically approached through the posterior cranial fossa. This exposure permits visualization of only the portions of the nerves extending across the posterior fossa and the medial meatus of the internal auditory canal.

The purpose of this paper is to present a middle cranial fossa surgical technique that allows visualization of the entire length of the internal auditory canal and the VII and VIII cranial nerves from the inner ear to the pons. This technique opens many possibilities of diagnosis and treatment of VII nerve, VIII nerve, and inner ear lesions.

Fourteen middle fossa internal auditory canal exposures have now been done by the author and Theodore Kurze, M.D., a neurosurgeon.

Description of Technique.

The operation is performed as a combined otologic-neurosurgical procedure. The neurosurgeon makes the incision, trephines the skull and closes the incision. The remainder of the surgery is performed by the otologist. The operative microscope is used for visualization and the diamond drill technique for bone removal. The incision used is shown in Figure 1.

A trephine opening about 4 cm. in diameter is made in the squamous portion of the temporal bone (see Fig. 2). This is

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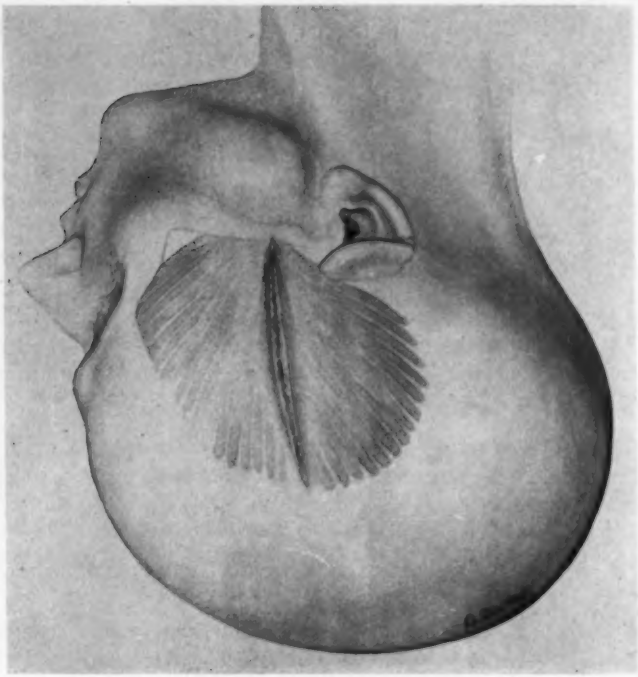

Fig. 1.

placed to allow exposure of the middle fossa surface of the petrous pyramid. Using the microscope at six-power, the temporal lobe is carefully elevated from the petrous pyramid by extradural dissection. Bleeding is minimized because magnification allows a very careful dissection. As the dural reflection is being carried medialward, the greater superficial petrosal nerve is encountered as it emerges from the petrous pyramid (see Fig. 3). This nerve is carefully exposed for a short distance.

The remainder of the dura is reflected from the arcuate eminence and back to the crest of the petrous pyramid. Diamond stones with continuous irrigation are used to remove the bone over the internal auditory canal. Diamond stones

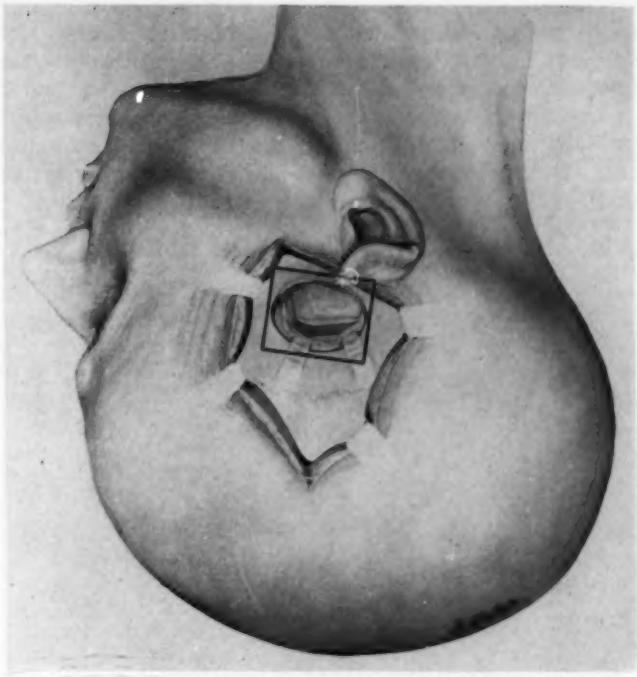

Fig. 2.

are considered essential since they do not tear the neurolemma. Continuous irrigation is essential for visualization by immediate removal of bone dust and to prevent thermal damage to the nerves and inner ear.

The bone removal is started at the point where the greater superficial petrosal nerve emerges from the petrous pyramid (see Fig. 4). This nerve is uncovered for one or two millimeters as it extends posteriorly to join the geniculate ganglion of the VII nerve.

The geniculate ganglion is then completely exposed, and the bone is removed over the VII nerve as it proceeds medialward to the internal auditory canal. As the internal auditory canal is approached, a blueness of the bone will develop, indi-

cating that the lateral end of this structure is near. It is important to remove only the bone superficial to the greater superficial petrosal nerve, the geniculate ganglion, and the VII nerve, because the cochlea is located in the bone anterior to the VII nerve and medial to the greater superficial petrosal nerve. The top of the vestibule and the superior semicircular canal are just posterior to the geniculate ganglion and the

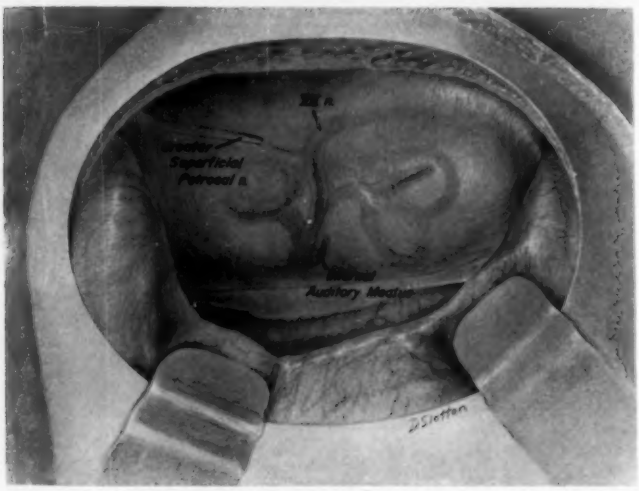

Fig. 3.

VII nerve. The cochlea and superior canal can be avoided by staying close to the VII nerve.

Once the internal auditory canal is reached, the cochlea and superior canal have been passed. The internal auditory canal is lined by an extension of the dura. This dura comes down around the VII nerve and becomes the neurolemma of that nerve. The dural cuff of the internal auditory canal is, therefore, exposed by removing the bone from the top half of the internal auditory canal. This dissection is carried medialward to the medial meatus of the internal auditory canal. Here the dura turns to form the posterior fossa dura covering

of the petrous pyramid. A small amount of this posterior fossa dura is exposed when the bone on the top half of the internal auditory canal is removed (see Fig. 5). The dura and the arachnoid of the internal auditory canal can now be opened along its entire length from the lateral termination of the canal to the posterior fossa dura (see Fig. 6).

This exposure allows the surgeon to visualize the VII nerve from the geniculate ganglion to the pons of the brain stem.

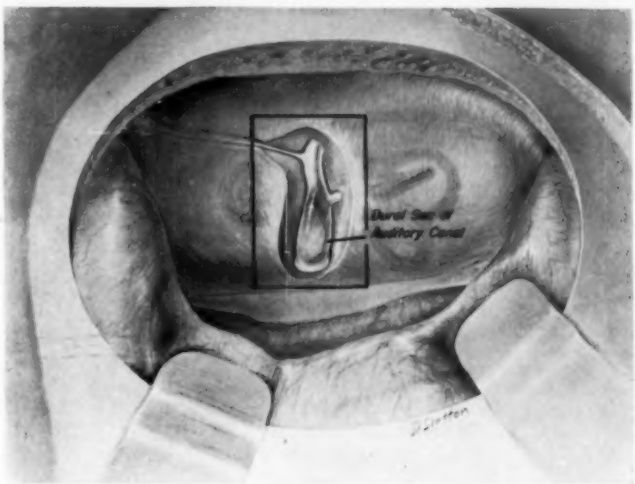

Fig. 4.

The VIII nerve can be seen from its entrance into the labyrinth to its origin in the pons. The internal auditory artery can be seen between these two nerves, and a portion of the cerebro-pontine cistern can be visualized.

Following any indicated surgical manipulation of the exposed structures, the wound is closed by placing gelfoam over the exposed internal auditory canal. The brain is allowed to re-expand and the incision is closed.

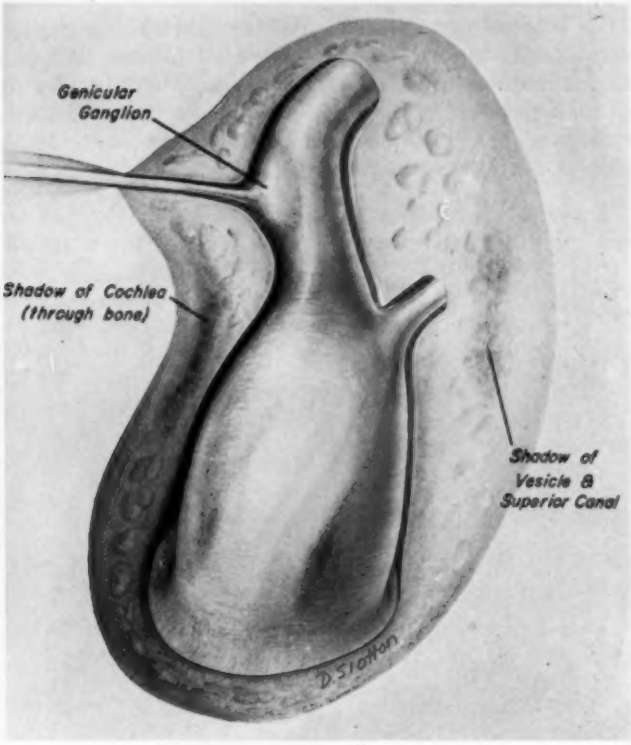

Fig. 5.

Postoperative Course.

To date, October, 1960, this approach has been performed 14 times. The first operation was performed on August 1, 1959, and the most recent one, two weeks ago. There have been no major postoperative complications in any of these patients. None of the patients has had postoperative facial weakness. During the first operation, the facial muscles repeatedly contracted spasmodically when the bone was being removed from over the geniculate ganglion.

One patient had a spinal type headache for one week which

was apparently due to loss of spinal fluid during the procedure.

One patient developed prostatitis on the sixth postoperative day.

The patients are kept in the hospital seven days postoperatively. They are ambulated on the third postoperative day.

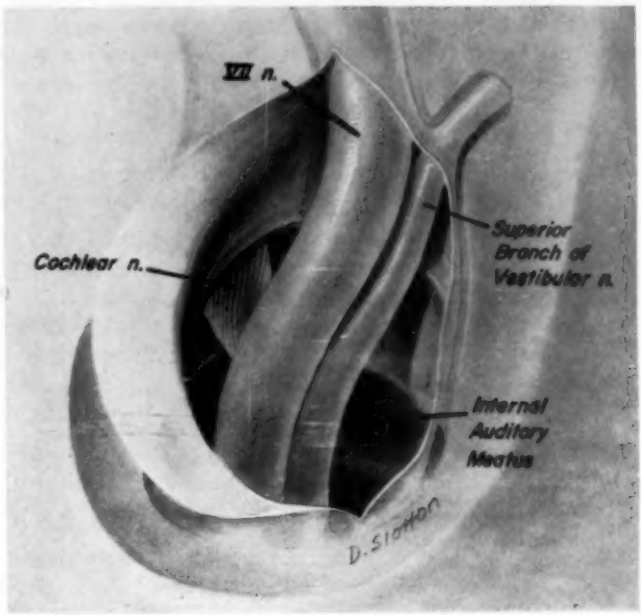

Fig. 6.

Clinical Applications.

The above approach should be useful in lesions involving the inner ear, VII and VIII nerves and the internal auditory canal vessels. To date, its greatest value has been in Ménière's disease. It has also been used in investigative surgery on otosclerotic inner ear deafness.

SURGICAL RELIEF OF VERTIGO BY SECTION OF THE VESTIBULAR NERVES IN THE INTERNAL AUDITORY CANAL.

The otologist is occasionally confronted with a patient in whom the differential diagnosis of early internal auditory canal tumor or Ménière's disease is a problem. Using the surgical approach described above, the internal auditory canal can be exposed and visualized. If a small tumor is present, it can be removed. If no tumor is found, a definitive diagnosis of Ménière's disease can be established. At the same time, the patient's vertigo can be relieved by sectioning the vestibular branches of the VIII nerve.

Discussion.

Hearing Loss. Hilger[1] states that the disease endolymphatic hydrops is first a cochlear disturbance and secondarily vestibular.

Pool and Pava,[2] in a review of 122 cases of acoustic neuroma proven by surgery, found that deafness preceded all other symptoms of acoustic neuroma in 90 cases (79 per cent) by an average of 3.9 years. In 16 of the remaining cases, other symptoms occurred simultaneously with the deafness. In only nine out of 122 cases did the deafness follow other symptoms.

In both Ménière's syndrome and acoustic neuroma, the hearing loss can be of gradual onset, sudden onset (Hallberg[3]), or fluctuating. In both conditions the hearing loss is of the sensorineural type and usually unilateral.

Fowler[4] in 1928, described recruitment. Dix, Hallpike and Hood[5] found loudness recruitment to be present in 30 cases of Ménière's disease. In 20 cases of VIII nerve degeneration due to neuroma it was absent. In six cases of cerebro-pontine angle tumor, recruitment was present but incomplete. The absence of recruitment in VIII nerve tumors is usually the rule, but exceptions are found so that this cannot always be depended upon.

Dix and Hallpike[6] later noted that in Ménière's syndrome with recruitment, discrimination was seriously impaired. In nerve deafness, on the other hand, ability to understand

speech increased with amplification up to a certain level of loudness, after which it maintained this degree of intelligibility or the intelligibility decreased slightly.

Schuknecht and Woellner,[7] however, found that in acoustic neuromas the loss of auditory discrimination was out of proportion to the loss for pure tone thresholds.

Tinnitus. Tinnitus usually accompanies all types of deafness. It can be the first symptom in both endolymphatic hydrops and VIII nerve neuroma. In Pool and Pava's[2] series of tumors, it was present in 51 per cent of the cases and in 7 per cent of the cases was the first symptom.

Vertigo. Vertigo can be the first symptom in both Ménière's syndrome and VIII nerve tumor. Wright[8] and Williams[9] each report that pseudo-Ménière's syndrome (vertigo without hearing loss) occurred in less than 10 per cent of each series of 500 cases. In Pool and Pava's[2] neuroma cases, vertigo was the initial symptom in 6.5 per cent and eventually occurred in 58 per cent of the cases.

There are no types of vertigo characteristic of either Ménière's or tumor. It can be spontaneous, postural, or initiated by movement. Most tumor cases complain of a constant unsteadiness not initiated by movement.[2]

There are no pathognomonic vestibular signs in these cases. Usually it is assumed that Ménière's syndrome has diminished caloric response, but complete loss or hyperactive responses can occur (Shambaugh[10]).

Out of 92 cases of tumor with reported vestibular tests studied by Pool and Pava,[2] one had a normal caloric response, three had minimal loss, 79 had moderate loss and nine had total loss on the ipsilateral side and some contralateral impairment.

X-ray. X-ray studies of the internal auditory canal should be made on all cases of unilateral hearing loss, tinnitus or vertigo. The literature on this point as reviewed by Pool and Pava[2] indicates reports of positive erosion of the internal

auditory canal in 50 to 90 per cent of cases. A normal roentgenogram does not rule out a tumor.

It is obvious, then, that there is no infallible way to differentiate early Ménière's syndrome from early acoustic neuroma. At present, the subsequent clinical course is the only suitable differential diagnostic method.

The next sign to develop after VIII nerve signs and symptoms in 85 per cent of Pool and Pava's[2] cases was V nerve sign of diminished corneal reflex. Headache, involvement of IX and X and cerebellar symptoms and signs develop several years after the initial symptoms. The VII nerve and the motor division of V are involved as a rule very late in the syndrome. Cushing[11] and others attributed this to the resistance of motor nerves to direct compression.

Case Report. Mr. B., a 57-year-old refinery worker from Houston, Texas, was referred to me by Dr. William K. Wright. He gave a history of increasing attacks of vertigo over a period of two and one-half years. Previous to surgery his attacks were coming three times a week and lasting several hours at a time. They were characterized by a sensation of the room's spinning from left to right. He had been unable to work for one year previous to surgery. He complained of tinnitus in his left ear which was increased during the vertiginous attacks. The patient's hearing had remained quite good, but for a few months previous to surgery, he had noted a slight loss of hearing in the left ear. He also had daily headaches located in the occipital region for several months prior to surgery.

Repeated pure tone audiometric studies in 1958 and 1959 revealed normal hearing except for slight bilateral sensorineural loss at 4,000 and 8,000 cps. There was some fluctuation of hearing, however, since an audiogram taken in January, 1959, and the day before surgery, showed a 42 db loss. Speech reception thresholds for the right ear were repeatedly 5 db and for the left, 15 db. The speech discrimination score in the right was 92 per cent and in the left, 88 per cent. The patient had no recruitment by equal loudness balance. The S.I.S.I. test was negative. A few months before surgery, however, the tone decay test indicated marked auditory fatigue in the left ear.

Caloric tests at first were normal, but in May of 1960, 30 seconds ice water syringing of the right ear resulted in two minutes 45 seconds nystagmus and only 45 seconds nystagmus from 30 seconds syringing of the left ear.

Neurological examination was carried out in 1958 and again in 1959. These revealed questionable left decreased corneal reflex. Skull films, mastoid films, encephalograms and spinal fluid examinations were all normal. There was no evidence of increased intracranial pressure. Just prior to surgery, a hyperesthesia of the first branch of the fifth nerve and decreased corneal reflex were noted on the left by one examiner, but not confirmed by another.

Medical therapy consisted of nicotinic acid, histamine and B-12. In

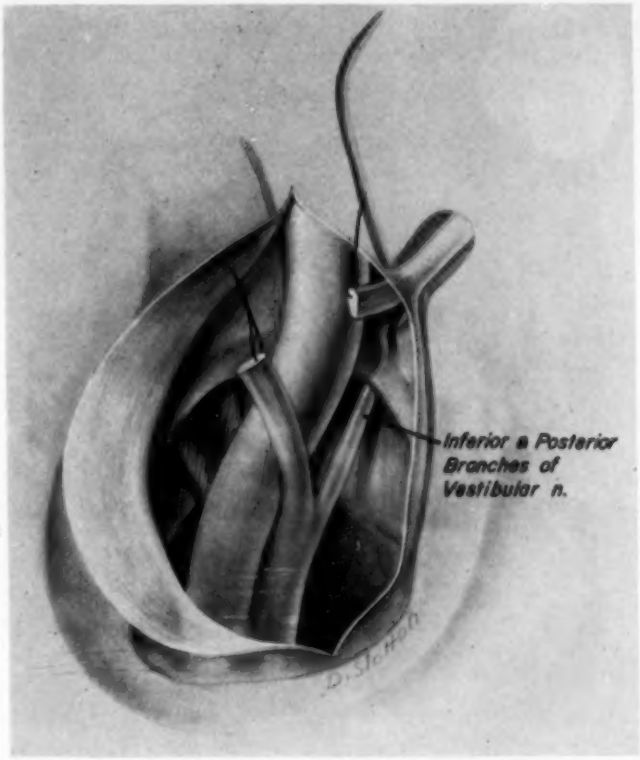

Fig. 7.

1959, he was given streptomycin, 1 gram twice daily for 44 days. There was no effect on his caloric or vestibular test.

In July, 1960, the left internal auditory canal was exposed through the middle cranial fossa. No tumor was found in the internal auditory canal; therefore, the superior and inferior vestibular branches of the VIII nerve were divided (see Figs. 7 and 8). Postoperatively the patient was slightly nauseated the first two days and had a fine nystagmus to the right. He was, however, able to retain food and fluids by mouth. On the third day, this nausea disappeared, and he was ambulated. He noted unsteadiness which completely disappeared by the tenth postoperative day. He has had no further attacks of vertigo. His hearing has remained at its preoperative level.

Preoperatively, a pulsating low-pitched tinnitus that increased with vertigo was present. This was gone following surgery and has not returned.

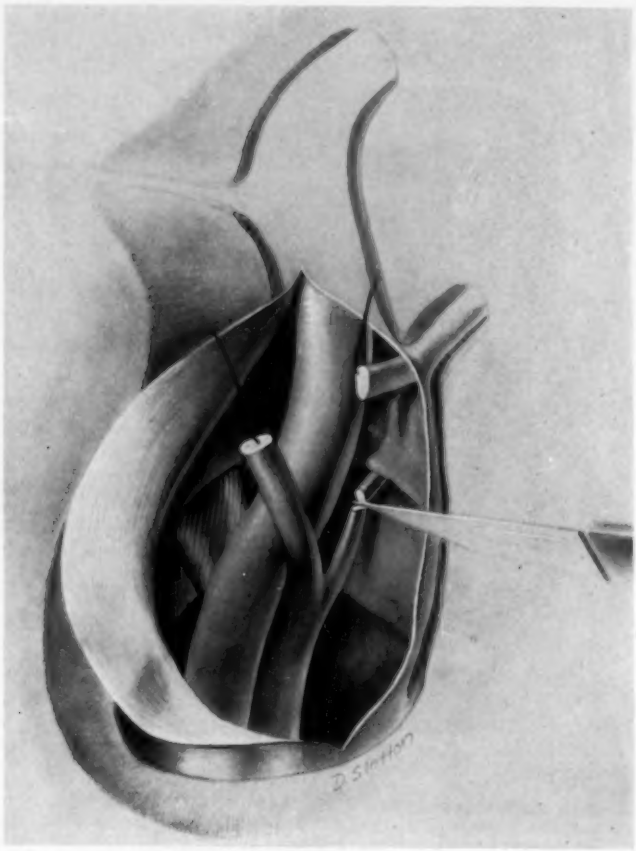

Fig. 8.

In summary, this patient had a clinical history of vertigo and tinnitus characteristic of either tumor or Ménière's disease. His lack of recruitment, and questionable V nerve findings pointed to a tumor. Normal neurologic findings including normal cerebrospinal fluid protein and normal pressure and normal X-ray findings suggested Ménière's disease.

In view of the incapacitating vertigo, something needed to

be done. Medical treatment had failed. A posterior fossa exploration and partial VIII nerve section as suggested by McKenzie[12] was a possibility. This approach, however, would not rule out the possibility of a small tumor in the internal auditcry canal. Neither would a destructive labyrinthectomy rule out the possibility of tumor and in addition, the patient would lose the hearing in the involved ear.

The middle fossa exploration and section of the vestibular nerves was an ideal approach to this patient's problem.

EARLY ACOUSTIC NEUROMA-VS-MÉNIÈRE'S SYNDROME.

The ideal time to remove an acoustic neuroma is when it is small and still confined to the internal auditory canal.

Hardy and Crowe[13] reported finding early asymptomatic acoustic neuromas in six out of 250 routine histologic temporal bone sections. Each of these six tumors arose in the medial end of the internal auditory canal; four from the vestibular portion of the nerve in the region of Scarpa's ganglia and two from the acoustic division opposite Scarpa's ganglia.

These tumors apparently grow very slowly and gradually increase the pressure in the internal auditory canal. This usually results in progressive unilateral sensorineural hearing loss that can precede other symptoms by many years.

Each of the tumors reported by Hardy and Crowe[13] was quite vascular. This would make it seem likely that thrombosis due to pressure could occur and cause marked fluctuation in symptoms or even sudden hearing loss as reported by Hallberg.[3]

At the present time, it is usually the practice to allow the tumor to grow until it extends into the cerebro-pontine angle before removing it. In this location the tumor becomes lethal and must be removed at all costs to save the patient's life. The cost is usually loss of the VII nerve and possibly the IX and X nerve and possible pyramidal tract deficits.

The operative mortality as reviewed by Pool and Pava[2]

varies from 2.4 per cent to 26 per cent in various series. The usual cause of death is hemorrhage into the brain stem, since the brain stem must be retracted in some cases to remove the tumor. Obviously, the smaller the tumor at the time of posterior fossa removal, the less morbidity and mortality. If, however, the patient is operated upon too early, the tumor will not have extended into the posterior fossa and, therefore, cannot be removed through this approach.

The approach presented above offers the possibility of removing these tumors while they are still confined to the internal auditory canal. The middle fossa approach has an almost negligible risk as compared with the posterior fossa approach, and offers the possibility of saving the VII nerve. This nerve is approached first in the middle fossa operation and blindly in the posterior fossa exploration.

If this concept of removal of very small acoustic neuromas through the middle fossa gains acceptance, a number of problems remain yet to be solved. One of these, which is being actively investigated on cadavers at present, is a surgical method of removing the petrous pyramid to allow the tumor to fall away safely from the brain stem and facilitate removal in case it has invaded the cerebro-pontine angle.

Another is to develop more accurate diagnostic methods to differentiate small tumors from other causes of sensorineural loss. Until such methods are developed in patients where a reasonable chance of early tumor is present, exploration of the internal auditory canal is indicated.

Case Report. Mrs. H., a 35-year-old stenographer, developed intermittent fullness and tinnitus in the right ear nine years before examination. Room spinning vertiginous attacks also occurred and became more frequent and more severe. Several courses of treatment with vasodilating medications, salt restriction and histamine were given.

Two years before surgery she was first seen in this office. Her hearing loss on the right at that time was 10 db. Her caloric was slightly diminished on the right. Extensive medical treatment including I.V. histamine and Cortisone was carried out. Severe attacks of vertigo, nausea, and vomiting continued to occur two or three times per month, and several attacks of unsteadiness occurred daily. Low pitched right sided tinnitus was present, and her hearing dropped to a level of 36 db throughout the speech frequencies on the right with a discrimination score of 76 per cent. Recruitment was present. X-rays taken one year apart were re-

ported by Dr. Compere to show possible enlargement of the right internal auditory canal.

In February, 1960, the right internal auditory canal was explored. No tumor was found in the internal auditory canal; therefore, the superior and inferior branches of the vestibular nerve were divided.

Except for some spinal type headache for one week, the patient made an uneventful recovery. She had a slight feeling of unsteadiness with sudden movement for two months postoperatively. Subjectively, the patient heard about the same. Pure tone testing revealed improvement for the speech frequencies from 47 db to 30 db. There was no response to ice water caloric stimulus in the right ear four months postoperatively. Her tinnitus was improved but was not totally absent.

Discussion.

This case was almost classic Ménière's disease except for questionable X-ray enlargement of the internal auditory canal.

Camp and Cilley[14] evaluated the measurements of 509 normal internal auditory canals. They encountered variations from 2.5 to 11 mm. on the two sides. Slight differences in the projection angle can give the appearance of changes in the internal auditory canal.

Discussion of Present Surgical Techniques for Ménière's Syndrome.

The most widely used surgical treatment for intractable Ménière's syndrome is labyrinthectomy. In the past, partial VIII nerve section was popular, but it has largely been abandoned because of the difficulties of the posterior cranial fossa approach.

Historical Background.

In 1904, Lake[15] removed the semicircular canals for Ménière's disease. Frazier[16] in 1908 sectioned the VIII nerve for "aural vertigo."

In 1932, McKenzie[17] performed the first partial VIII nerve section to divide the vestibular portion of the VIII nerve. A large series of these operations was done by McKenzie[17] and Dandy.[18] The principal advantage of this operation was that it did not destroy the hearing remaining in the affected ear.

During the 1930's and 1940's the number of cases of Ménière's disease coming to surgery was greatly reduced by the introduction of medical treatment.

In 1939, Day[19] attempted to retain hearing by a partial electro-coagulation of the labyrinth. He concluded, however, that usually the hearing should not be saved in the affected ear, because poor discrimination and recruitment made the remaining hearing annoying to the patient. The lack of desire to salvage the hearing and unwillingness to subject the patient to posterior fossa exploration has led to almost total abandonment of partial VIII nerve section.

Today most otologists treat Ménière's disease with various medical regimens. In most instances the vertigo is relieved, but usually the tinnitus and hearing loss persist. If the vertigo becomes incapacitating in spite of treatment, a labyrinthectomy is recommended. This is an ideal solution if the patient's hearing loss has deteriorated below useful levels (usually assumed to be less than 50 db average for the three speech frequencies, 500, 1,000, 2,000 cps.).

Otologists continue to see patients who, in spite of adequate medical treatment, have very annoying or incapacitating vertigo, but who have hearing that is useful and valuable in the affected ear. To help this group of patients, in 1927, Portmann[20] recommended drainage of the endolymph through the saccus endolymphaticus. Passe[21] has performed cervical sympathectomies on these patients. Each of these procedures has been reported to give good results (Flett[22] and Passe[21]), but both need wider clinical trial to be proven.

Medical destruction of the labyrinth with streptomycin has been recommended and reported extensively by Schuknecht.[23]

Surgical exposure of the otic capsule and destruction of part of the labyrinth with ultra sound has been recommended by Arslan[24] and is being given clinical trial at the present time.

Results of Partial Nerve Section in Ménière's Disease.

Barber and Ireland[25] reported a long term follow-up of 117 patients who had had partial section of the vestibular portion of the VIII nerve by McKenzie between 1931 and 1951. Eighty-three per cent had total permanent relief of

vertigo. Four per cent had partial relief, and 13 per cent had no relief.

One-third of these patients retained useful hearing (above 50 db by SRT) for ten or more years. Two-thirds of the patients had gradual decline of hearing over a period of ten years. Complications were two deaths from meningitis and one partial facial weakness.

Green[26] reported on the long term follow-up of 401 out of 587 of Dandy's cases of nerve section. Total relief of vertigo with partial or complete section of the VIII nerve resulted in 90 per cent of the cases. Two deaths were reported from meningitis, and permanent facial paralysis occurred in 1.7 per cent of the partial section cases and in 9.2 per cent of the total section cases.

In summary it seems that section of the vestibular portion of the VIII nerve gives 90 per cent chance of permanent relief of vertigo. Most of the recurrent cases of vertigo represent bilateral Ménière's disease patients. One-third of these patients will retain useful hearing on the affected side, and two-thirds will have continued gradual deterioration of hearing as a result of their disease.

The objections to the use of this surgery would, therefore, seem to be the magnitude of the surgical procedure of posterior fossa exploration and not poor results.

THOUGHTS ON THE POSSIBLE SURGICAL RELIEF OF TINNITUS.

Tinnitus remains today as one of our great otologic mysteries. It can be started by such diverse causes as exposure to loud sound; changes of middle ear pressure; otitis media; otosclerosis; quinine, streptomycin, or aspirin therapy; VIII nerve neuritis; Ménière's disease; and VIII nerve tumors.

These causes are known to affect the end organ mechanism. It seems most likely, therefore, that tinnitus represents a stimulus arising in the cochlear or vestibular labyrinth rather than in the central pathways of hearing.

Assuming that tinnitus arises peripherally, the nerve path-

way would be through the internal auditory canal to the central nervous system where the tinnitus is perceived. The VII or VIII nerve or both must, therefore, conduct the tinnitus stimulus.

Tinnitus was completely relieved in 37 per cent of the total VIII nerve section, and 28 per cent of the partial VIII nerve section cases of Dandy.[26] Eight per cent of 52 cases of labyrinthectomy performed through a fenestra in the horizontal canal reported by Cawthorne[27] had total relief of tinnitus. Simonton[28] reported loss of tinnitus in 16 per cent of 97 patients following labyrinthectomy. Lempert[29] stated that labyrinthectomy through the horizontal canal only occasionally resulted in loss of tinnitus. He, therefore, recommended a decompression operation consisting of transcanal removal of the stapes and opening of the round window with a burr. This he felt allowed for a more complete degeneration of the cochlea and less inflammation of the cochlear nerve. He reported ten cases operated upon in this way with relief of tinnitus in nine.

Rosen[30] points out that in lower animals, hearing is transmitted through the VII and IX nerves. Electrical stimulation of the chorda tympani can produce tinnitus. Inflammation of the geniculate ganglion in Ramsey-Hunt syndrome often produces tinnitus.

The above material indicates that in approximately one-third of the patients with Ménière's disease, tinnitus can be relieved by partial or total VIII nerve section; therefore, it seems likely that in the other two-thirds, the tinnitus could be mediated through the VII nerve.

Within the internal auditory canal, there is a connection between the VII and VIII nerves. Shute[31] describes the facio-cochlear anastomosis as being sensory with cell bodies in the geniculate ganglion and the fibers extending down to the cochlear portion of the VIII nerve. This facio-cochlear anastomosis could conceivably be a nerve pathway for tinnitus.

Case Report. Mr. A., a 51-year-old barber, presented with recurrent incapacitating vertigo for two years. This was accompanied by a right 32 db sensorineural hearing loss with 76 per cent discrimination. Recruit-

ment was present in the right ear. He also complained of marked fluctuating low-pitched tinnitus. This was increased during his attacks.

Despite adequate medical therapy, the patient remained incapacitated by the vertigo. Caloric tests were normal, and petrous pyramid X-rays showed no abnormality.

The preoperative diagnosis was Ménière's disease. Because of his vertigo, either labyrinthectomy or vestibular nerve section was indicated. The vestibular nerve section was chosen because there was a better chance of saving the remaining hearing and also a better chance of relieving the tinnitus. In May of 1960, this procedure was carried out.

At surgery the VII nerve and the vestibular portions of the VIII nerve were well visualized through the middle cranial fossa. A blunt hook was passed between the VII and VIII nerves in an effort to divide the facio-cochlear anastomosis. As this was being done, several capillary loops lying on the VIII nerve were torn. The superior and inferior divisions of the vestibular portion of the VIII nerve were divided, and the wound was closed.

Postoperatively, the patient made an excellent recovery. No facial weakness was present, the tinnitus was gone, and in three days he was ambulating with minimal unsteadiness. By gross speech testing through the bandages in the hospital, the patient felt his hearing was unchanged on the operated side.

Nine days postoperatively, a pure tone audiogram revealed total hearing loss on the right. This has persisted. The patient is pleased with his result, however, because he has returned to work and no longer has tinnitus. He is not particularly aware of his further hearing loss.

Comment.

Refinement of the surgical technique will make it possible to divide the facio-cochlear anastomosis without tearing the delicate branches of the internal auditory artery.

The two cases presented above also had an attempt made to divide the facio-cochlear anastomosis. This was done with a small right angle footplate hook. These delicate nerves are not easy to see and definitely divide, but improvement of technique should make this surgery easier.

One other patient with severe vertigo and good hearing has been operated upon. At the time of this writing, she is only ten days postoperative, but is feeling fine and is free of vertigo. Her tinnitus was never marked, but seems to be improved.

SURGERY FOR VIII NERVE COMPRESSION.

The following case report illustrates benign VIII nerve compression with progressive hearing loss.

Case Report. Miss M., a 57-year-old secretary, experienced the onset of left sided tinnitus two and one-half years before surgery. This had disappeared, but returned in six months accompanied by sensorineural hearing loss of about 20 db. X-rays by Dr. Compere revealed a large radiolucent area in the left petrous pyramid superior to the internal auditory canal extending from the superior semicircular canal to the petrous tip. The internal auditory canals appeared equal bilaterally. The possibility of a large petrous air cell unrelated to her symptoms seemed the most likely reason for the X-ray finding.

Under continued observation and vasodilating medication, the patient's unilateral hearing loss and tinnitus increased in severity. At no time did the patient experience vertigo.

Eighteen months following the first X-ray examination, petrous pyramid X-rays showed a definite enlargement of the radiolucent area superior to the internal auditory canal. The hearing had now dropped to an 80 db level with no discrimination. Caloric response was depressed in the left ear.

The possibility of a primary cholesteatoma (no previous history of ear infection) or some unusual tumor was strongly considered. A middle fossa, petrous pyramid exploration was recommended.

At surgery in January, 1960, a large petrous air cell was encountered superior to the internal auditory canal. The bone was removed from the superior half of the internal auditory canal using the above described approach. There seemed to be some narrowing of the canal by extradural bony constriction at the junction of the medial third and lateral two-thirds. This bony constriction was removed, and the dural sac lining the internal auditory canal was opened. The VIII nerve was found to be under pressure and ballooned out of the canal so that the edges of the dural sac could not be re-approximated. The area was covered with gelfoam, the temporal lobe was allowed to re-expand and the incision was closed in the usual fashion.

Postoperatively the patient's hearing did not continue to deteriorate but has actually improved by 12 db. Discrimination is still poor and the tinnitus is unchanged.

Comment.

Several cases demonstrating large air cells in patients with progressive sensorineural hearing loss have been noted by Dr. Compere,[32] on X-ray examination. This is the first case followed carefully enough to note enlargement. It is difficult to say whether the enlarging cell is the etiology of this patient's hearing loss, or if this represents a unilateral VIII nerve neuritis. If such cases are explored in the future, possibly biopsy of the vestibular portion of the VIII nerve would help establish the diagnosis and suggest possible treatment.

VIII NERVE COMPRESSION BY OTOSCLEROSIS.

This paper is intended to present a surgical approach to

the internal auditory canal and its contents. I do not wish, therefore, to present a lengthy discussion of the reasoning behind the decompression of the internal auditory canal in otosclerosis. A paper on this subject together with case reports is now being prepared.

Suffice it to say that a number of authors have postulated from histologic sections that the VIII nerve can become compressed by otosclerotic foci. These otosclerotic internal auditory canal compressions have been demonstrated a number of times by X-ray as reported by Compere.[33]

Eight patients with far advanced sensorineural hearing loss from otosclerosis have undergone internal auditory canal decompression. The hearing improvement from this procedure to date has not been promising. These cases are mentioned here because none of them have had any postoperative facial weakness or intracranial complication as a result of internal auditory canal exploration through the middle cranial fossa.

FACIAL NERVE LESIONS OF THE INTERNAL AUDITORY CANAL.

The middle fossa approach to the internal auditory canal opens up the possibility of treating certain lesions of the facial nerve.

Two weeks before writing this paper, a large neurofibroma originating in the geniculate ganglion of the facial nerve of a four-year-old child was removed. A detailed case report of this patient is to be prepared when sufficient time has elapsed for adequate follow-up.

The exposure obtained by this surgical procedure suggests the possibility that a facial nerve torn by fracture of the petrous portion of the temporal bone could be repaired through the middle fossa approach.

CONCLUSIONS.

1. A new surgical method of exposing the contents of the internal auditory canal through the middle cranial fossa is presented.

2. This approach has been used for various lesions on 14 patients. There have been no postoperative facial weaknesses or intracranial complications in these patients.

3. This approach has proved to be valuable for sectioning the vestibular branches of the VIII nerve.

4. Speculation as to other uses of this approach is presented.

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SCLEROMA IN A NONENDEMIC AREA.

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Scleroma, a chronic granulomatous affliction of the respiratory tract, is encountered rarely in natives of the United States, particularly those of the Midwest. In recent years two brothers of Mexican extraction with the clinical and histopathological features of scleroma have been followed in the University Hospitals at Iowa City. One was born in Iowa, and both had resided in the state most of their lives. The rareness of this unusual granuloma and the difficulties encountered in establishing the diagnosis in a nonendemic area prompted the authors to report their experiences.

Scleroma in Two Residents of Iowa.

Case 1. The older brother (L.B.) was first seen in July, 1955, at age 18 with complaints of nasal obstruction, persistent hoarseness and increasing amounts of "thick, yellow phlegm in my throat." Except for the first three years of his life spent in Texas, where he was born, his only excursion from the state was for a year in Michigan while in the Army.

The initial examination revealed a young adult male of Mexican extraction with an enlarged, broadened nose and a severely stenotic left nasal cavity. The faucial tonsils were absent and there were prominent islands of lymphoid granulations on the posterior pharyngeal wall. The nasopharynx was narrowed laterally, rendering mirror examination unsatisfactory. The entire larynx was chronically reddened, and the true cords appeared thickened. The anterior half of the larynx was poorly visualized because of an overhanging epiglottis. X-rays of the paranasal sinuses and chest were normal.

The finding of a scarred, warped nasal septum provided an obvious explanation for his nasal obstruction and was consistent with a history of repeated boxing trauma over the preceding two years. He was admitted to the hospital to undergo a nasal septal reconstruction. Attempts to pass an endotracheal anesthesia cannula were blocked by a firm, nonulcerated mass arising from the anterior tracheal wall in the immediate subglottic area. Acute respiratory obstruction ensued, requiring tracheotomy and cancellation of the proposed nasal surgery. Subsequently, this mass was excised subtotally under suspension laryngoscopy (see Fig. 1). Histologically it possessed features consistent with but not diagnostic of scleroma. Biopsies of the nasal mucosa showed the same histological

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changes. He was decannulated and discharged from the hospital. He failed to return for further examination until November, 1959, at which time his brother was also examined.

In the intervening four years his symptoms of hoarseness and slight exertional dyspnea had persisted unchanged, but he was able to work as a railroad laborer and occasionally boxed professionally. He had received no treatment for scleroma nor had he received antibiotics for other infections. His symptoms of cough and hoarseness were unchanged. The external appearance of his nose was unchanged when compared with photographs taken during his hospitalization in 1955. The nasal septum

Fig. 1. Scleroma in the subglottic area. Larynx of L.B. (Case 1) as seen at direct laryngoscopy. Scarring in the aryepiglottic folds causes the epiglottis to overhang, obscuring the anterior portion of the glottis.

was markedly deviated to the left and dislocated off the palatine crest. Smooth white nodularities on the septum and turbinates bilaterally contributed to his nasal obstruction. The nasopharynx was narrowed by scarring laterally. Laryngeal examination showed crusted, irregular, granular areas in the posterior commissure and left subglottic area. On direct examination scarring in the areas lateral to the arytenoids was observed. This prevented the posterior ends of the vocal cords from approximating on phonation. Bronchoscopy revealed the remainder of the tracheobronchial tree to be normal.

Repeated cultures of the nose, nasopharynx and trachea recovered a mixed flora but no *Klebsiella rhinoscleromatis*. Complement fixation tests using the patient's serum and known *K. rhinoscleromatis* did not show complement fixation above a 1:4 dilution of serum and were considered negative. Biopsies of the subglottic, interarytenoid area, and nasal nodules showed dense fibrosis with a chronic inflammatory response. *K. rhinoscleromatis* was not recovered in cultures of ground tissue removed from these sites.

This patient has been followed regularly since February, 1959. During this period he completed three four-week courses of tetracycline, receiving a gram a day. There has been no change in the physical findings. He has continued to be hoarse and to breathe through his nose with difficulty, but otherwise is in good health.

Case 2. The younger brother (F.B.) was referred to the University Hospitals in February, 1959, at the age of 18, with a nine-month history of voice weakness and increasing hoarseness. He was born in Iowa, and his only trip out of the state was for a three-month visit with relatives in Texas one year prior to initial examination.

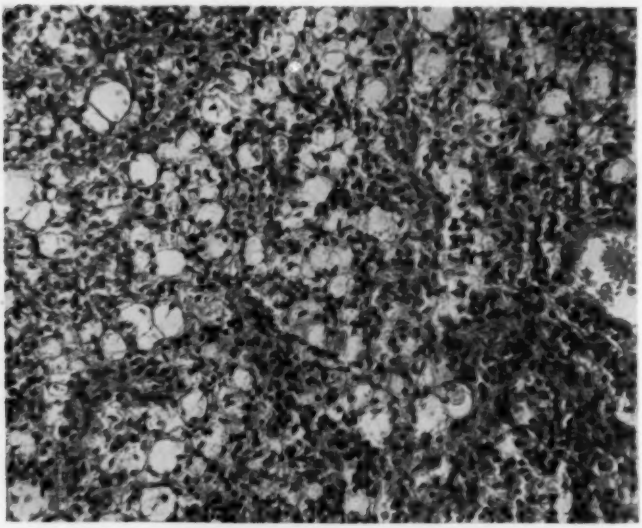

Fig. 2. Florid Scleroma. Biopsy of a red granular mass in nasopharynx of F.B. (Case 2). The large cells with almost clear cytoplasm except for lacy strands are Mikulicz's cells. Gram-negative rod bacteria were found in these cells using special staining techniques. The smaller cells are predominately plasma cells. H&E. x210.

His external nose appeared normal. Yellow purulent material filled both nostrils. The nasal mucosa was hyperemic, granular and bled easily with slight trauma. It was heaped in several nonulcerated mounds on the septum and turbinates. A firm, red, granular mass extended to the left lateral nasopharyngeal wall from the soft palate. The soft palate was retracted upward and to the left with disappearance of the uvula. Although the faucial tonsils had been removed previously, hyperemic, granular tissue occupied the superior pole of the left tonsillar fossa. There were hyperemic granular areas on the laryngeal surface of the epiglottis, in the interarytenoid area, on the aryepiglottic folds and in the immediate subglottic region. Crusts and yellow purulent material ad-

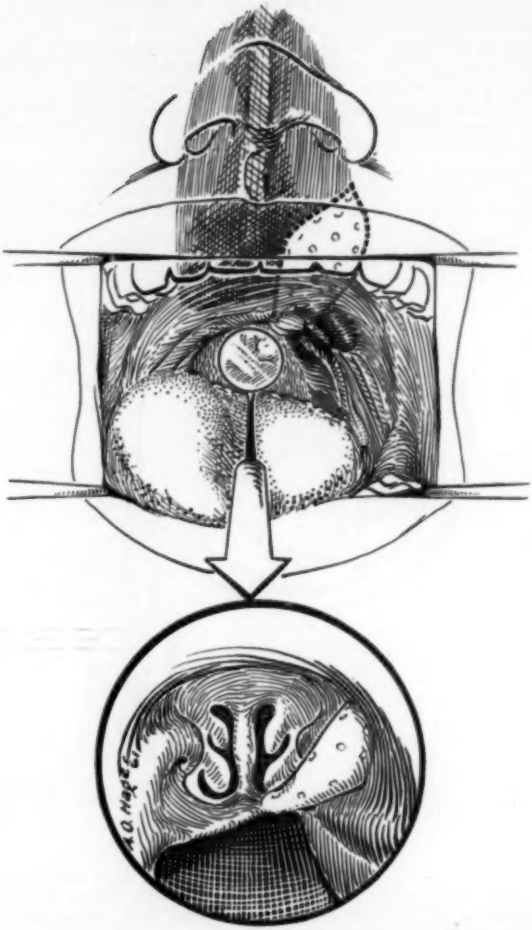

Fig. 3. Scleroma after treatment. Pharynx and nasopharynx of F.B. (Case 2) after 30 days of treatment with tetracycline, redrawn from a photograph. The soft palate is scarred and the uvula is not seen. The red granular mass extending from the left lateral pharyngeal wall to the palate when first seen has been converted to a glistening white, firm band.

hered to these areas and to the true cords, which were slightly hyperemic and edematous but otherwise uninvolved.

Biopsies of the granular areas in the nose, nasopharynx and larynx possessed the histologic features of florid scleroma including Mikulicz's cells, plasma cell exudate, Russell's bodies, hyalinized vessels and squamous metaplasia of the epithelium (see Fig. 2). Faintly staining Gram-negative rods were found in the Mikulicz cells, and *K. rhinoscleromatis* was recovered from the ground biopsy tissue.

An encapsulated Gram-negative rod possessing the biochemical characteristics of *Klebsiella rhinoscleromatis*, was recovered from cultures of the nasal discharge (see Table I). Dr. Phillip Edwards[a] determined

Fig. 4. Hamartoma, composed of fibrous and vascular elements, in the subglottic space of a 7-year-old girl, causing respiratory obstruction. a. Appearance of mass at direct laryngoscopy.

the O&K antigen of this organism, finding it to have an O:2 and K:3 antigen, thus confirming the identity of this organism.[b] It was found to be sensitive to tetracycline, streptomycin and chloromycetin, and resistant to penicillin and sulfonamides.

Egg inoculations with a supernate of the ground biopsy tissue failed to show viral growth. Complement fixation tests were performed using the patient's serum and the recovered organism. The serum was held constant at a 1:8 dilution. 4+ reactions were noted at a 1:32 dilution

a. Chief, Enteric Bacteriologic Laboratory, Communicable Disease Center, Atlanta, Georgia.

b. For a list of the biochemical tests used in the identification, see Table I.

TABLE I.

Criteria used in identification of organism in *Case 2* (F.B.) as *Klebsiella rhinoscleromatis;* (9,20,25,34,38) characteristics ascribed to *Klebsiella rhinoscleromatis;* and a typical strain of *Klebsiella pneumoniae* listed for comparison.

A = Acid only. A&G = Acid & Gas.
A± = Acid only, slowly if at all. V = Variable.

	Typical Klebsiella pneumoniae	Klebsiella rhinoscleromatis	Case 2 (F.B.) Organism	K. rhinosci. Levine-Hoyt Criteria (25)
Gram Stain	Neg	Neg	Neg	Neg
Morphology	Rod	Rod	Rod	Rod
Capsule	+,-	+	+	+
Large, Mucoid, Coalescent Colonies	+	+	+	+
Aerobic	+	+	+	+
Motility	-	-	-	
Voges-Proskauer (Acetylmethyl-carbinol)	+	-	-	
Methyl Red	-	+	+	
Indole	-	-	-	-
Nitrate	+	+	+	+
Citrate	+	-	-	
Urea	+	-	-	
Glucose	A&G	A	A	A
Lactose	A&G	-	-	-
Maltose	A&G	A	A	A
Mannite	A&G	A	A	A
Sucrose	A&G	A	-	A±
Xylose	A&G	A	A	
Salicin	+	+	+	
Inositol	+	+	+	
Adonitol	+	+	+	
Dulcitol	V	-		
d-Tartrate	+	-	-	
H_2S	-	-	-	
Gelatin	-	-	-	
Mucate		-	-	
O Antigen	1-5	2(2A)	2	
K Antigen	1-72	3	3	

of the antigen; 1+ at 1:64. The antigen was anticomplementary at 1:4 but not at 1:8. The serum was not anticomplementary.[c]

Having confirmed the clinical impression of scleroma by biopsy, culture and a positive complement fixation test, tetracycline, [illegible] grams a day, was started. After five days of this therapy the patient developed sore throat, odynophagia, fever up to 102.4°, and a green, watery diarrhea. A hemolytic *Staphylococcus aureus* was cultured from his pharynx. The tetracycline was stopped and these symptoms subsided promptly. Tetracycline,

c. All of the complement fixation tests and virus studies in this report were conducted by Dr. Albert McKee, Department of Bacteriology, Medical Laboratories, Iowa City, Iowa.

Fig. 4. b. Section of mass showing its covering of hypertrophied squamous epithelium and relatively narrow base.

a gram a day, was resumed ten days later and was well tolerated. The redness and granularity of the respiratory passages subsided dramatically with this therapy. He was discharged from the hospital on tetracycline for 30 days.

One month after discharge, a white, firm scar had replaced the hyperemic granular nodules. The nose and larynx were crusted, and his hoarseness was still present. *K. rhinoscleromatis* was not recovered from cultures of the nose and pharynx. Six months later, after a second course of tetracycline, his hoarseness had disappeared, and his nasal obstruction was greatly relieved. The nasal mucosa was less crusted, and the larynx was normal. Direct laryngoscopy and bronchoscopy revealed a normal larynx and tracheobronchial tree except for a small, crusted, scarred area in the right subglottic region. The mass in the nasopharynx, now a glistening white mulberry scar, was biopsied, and microscopically was found to be composed of dense fibrous tissue containing scattered chronic

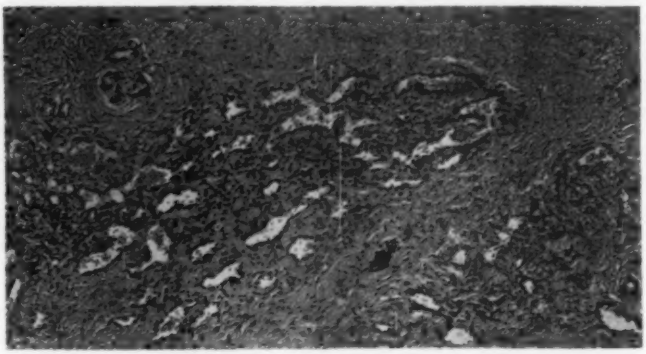

Fig. 4. c. Maze of thin-walled vascular channels prominent throughout this tumor.

inflammatory cells without the specific features of florid scleroma. Cultures of the nasal secretions, tracheal aspirations and ground biopsy tissue grew out normal flora.

Complement fixation tests were used using a 10^{-4} suspension of ground tissue excised from the nasopharynx as antigen and a 1:8 dilution of the patient's serum as antibody fixed complement at a 1:2 and a 1:4 dilution of the 10^{-4} tissue suspension. This was interpreted as good evidence that this patient had antibodies against an antigen in the tissue excised.

He was given an additional 30 days of tetracycline, and was asymptomatic when examined in March, 1960, 12 months after the diagnosis had been established; his cough had disappeared and the hoarseness had not returned. Physical examination was unchanged except that the nose was no longer crusted. Cultures showed only normal flora. Complement fixation tests using the patient's serum and the organism isolated from his nose 13 months before did not fix complement in dilutions of serum above 1:4 and were, therefore, considered negative.

COMMENT.

The clinical impression of scleroma was confirmed by bacteriologic, serologic and histopathologic evidence in Case 2, but not in Case 1. The symptoms and physical findings were dramatically altered by specific antibiotic therapy in Case 2 but were unchanged in Case 1. This should not be surprising, for while both of these patients' presenting clinical features were those suggesting scleroma initially, they were examined in different phases of the disease. F.B. (Case 2) when first seen was in the florid, granulomatous stage while his older brother presented in the scarred end-stage of "burned-out" scleroma.

The fall of the complement fixation titer in Case 1 from a significantly high to a non-specific low titer after treatment suggests an explanation for the insignificant titer found in Case 1.

The dramatic improvement in Case 2 with specific antibiotic treatment paralleled by the disappearance of *K. rhinoscleromatis* from the respiratory tract and the drop in complement fixation titer supports the thesis that *K. rhinoscleromatis* is an etiologic agent in this disease.

The deaths of the father, mother and two brothers from accident and causes other than scleroma limited the possibility of an epidermological study. The patients' only living siblings, two sisters, were alive and apparently well, but unavailable for study, as were the relatives in Texas.

Scleroma-Like Processes.

Alerted by the classic clinical, histopathological, bacteriologic and serologic findings demonstrated in Case 2, other patients were examined more closely with this entity in mind. Our experiences with two patients with scleroma-like lesions illustrate some of the problems of differential diagnosis, particularly in a non-endemic area.

Case 3. J.C., a seven-year-old girl of Mexican lineage from the same city as the brothers, Cases 1 and 2, was referred to the hospital because it was not possible to decannulate her two weeks after tracheotomy performed for acute respiratory obstruction, presumably the result of acute laryngo-tracheo-bronchitis. Past history revealed that her first

three postnatal days were complicated by episodes of cyanosis with mass clonic movements usually associated with feeding. From infancy to the time of admission here in April, 1958, at age six years, she had been a sickly child, who had repeated, prolonged, respiratory infections characterized by nasal discharge, cough, hoarseness and fever. She was hospitalized for pneumonia on three occasions and had received numerous courses of antibiotics.

Examination at admission in April, 1958, revealed a thin dark-skinned female with a tracheotomy cannula in place. She was afebrile; routine blood counts were normal. She spoke in a hoarse whisper and developed acute respiratory obstruction if the tracheotomy cannula were corked, despite replacing the cannula with one of smaller diameter. Hyperemia and slight edema of the entire larynx were the only other abnormalities detected by mirror examination. By suspension laryngoscopy a pink, smooth, firm pedunculated lesion was seen in the immediate subglottic area, filling most of the lumen of the trachea. This mass arose from the posterior and right lateral wall of the trachea. It was subtotally removed. Its appearance and histological structure is illustrated in Figure 4.

The patient was decannulated and discharged, remaining well for 14 months, until August, 1959, when increasing respiratory obstruction again occurred, necessitating tracheotomy. A bronchoscopy four months earlier had revealed a good airway and no structural abnormalities. The absence of fever and normal blood count on admission did not confirm the admission diagnosis of acute laryngo-tracheo-bronchitis. A smooth, pedunculated, white, glistening mass in the immediate subglottic area was visualized by indirect laryngoscopy. This was subtotally resected endoscopically in two stages. It was identical histologically to the tumor removed from this same area 15 months earlier. Laryngoscopy and bronchoscopy in April, 1960, revealed only a slightly raised mound at the site of the base of the lesion previously resected. No other abnormalities were found in the upper respiratory tract except small, cryptic faucial tonsils.

Repeated cultures of this girl's nasal and bronchial secretions, ground cultures of the faucial tonsils, adenoids, and from the subglottic mass, were consistently negative for *K. rhinoscleromatis*. Complement-fixation tests against known *K. rhinoscleromatis* were consistently negative. She is being observed at four-month intervals and is asymptomatic at the time of writing.

COMMENT.

In view of the large prominent vascular channels near the surface and relatively sparse chronic inflammatory exudate, the histology of the resected lesions is more suggestive of a hamartoma composed of vascular and connective tissue than an inflammatory process. Its recurrence 15 months later with the identical microscopic picture supports this impression. This lesion may represent a partially involuted subglottic hemangioma, possibly present before birth and hence accounting for her stormy immediate postnatal course and subsequent respiratory difficulties.

Case 4. J.F., a 36-year-old Caucasian male was an extremely poor historian who presented a long history of mental disturbance. He had been a resident of Iowa all his life. He performed odd jobs and was in apparent good health until he developed ankle edema following a sore throat in 1958. He was first seen at University Hospitals in May, 1959, at which time the diagnoses of crossed, ectopic right kidney, chronic pyelonephritis and hypochromic anemia were made.

Purulent nasal discharge was noted by an examiner in October, 1959, although the patient consistently denied any symptoms referable to his nose or throat. Anterior rhinoscopy revealed a smooth, 2x1x1 cm. mass attached by a broad base to the floor and the base of the septum in the left nostril. This was smooth, covered by mucosa and firm to touch. Another 5 mm. nodule was noted high up on the left side of the septum posteriorly. The turbinates were moderately hypertrophied and there was a large amount of thick yellow pus in both nostrils. The nasopharynx, pharynx and larynx were normal. X-rays of the paranasal sinuses showed opacity of all the paranasal sinuses with an air-fluid level in the left maxillary sinus. Pus was obtained by irrigation of both maxillary antra.

Cultures of the antral washings produced a mixed flora of *micrococcus, alpha hemolytic Streptococcus, E. Coli, Staphylococcus epidermitis, hemolytic Staphylococcus* and *Pseudomonas aergeniosa.* No Klebsiella-like organisms were obtained from repeated cultures. Of these organisms found, *E. Coli* and *hemolytic Staphylococcus aureus* were persistent and were recovered consistently from the nasal secretions, from the antra when Caldwell-Lucs were performed, and from the ground tissue removed from the mass in the floor of the left nostril. Microscopic examination of the tissue taken from the sinus showed fibrovascular proliferation accompanied by infiltration with chronic inflammatory cells, particularly plasma cells. The tumor-like mass in the left nostril was histologically similar except that several focal aggregates (usually located in the immediate submucosa) of large cells with central nuclei and foamy cytoplasm occasionally containing a few gram-negative rods were present but not seen in the tissue removed from the antra.

This patient's serum failed to fix complement using known *K. rhinoscleromatis* as antigen. Histoplasmin, tuberculin, and serology tests were negative.

Allergy studies demonstrated evidence of dust and mold sensitivities. A desensitization program to these inhalants was started.

Since Caldwell-Luc sinusotomies performed in December, 1959, this man's purulent nasal discharge has subsided, and both antra wash clear. On physical examination the nose, nasopharynx, pharynx and larynx were normal at the last examination in December, 1960. At the site of the previous mass in the left nostril, only a 5 mm., smooth, mucosa-covered mound remained. The lesion seen previously high posteriorly on the left side of the septum was no longer visualized. Only "normal flora" was recovered from the nasal secretions.

COMMENT.

This case illustrates that the individual histologic features of scleroma are nonspecific and that the diagnosis of scleroma, at least in nonendemic areas, should be supported by clinical,

histological and laboratory evidence, if the term is to be useful in defining a specific disease entity.

Cases with atypical features such as this might be labeled "scleroma-like processes" to avoid the confusion which results when these cases are grouped with those in which the clinical, histological and laboratory criteria of typical scleroma are satisfied.

DIFFERENTIAL DIAGNOSIS.

Criteria used in establishing the diagnosis of scleroma can be grouped into the following categories: *1.* clinical; *2.* histopathological; *3.* bacteriological; and *4.* serological. The criteria which are used necessarily will reflect one's definition of this entity and his views as to its etiology.

1. Clinical Criteria.

Descriptions of the clinical features of scleroma in its various states are remarkably consistent.[4,5,8,10,16,25,27,29,30,31,32,33,39] In a scleroma-endemic area, clinicians have used physical findings alone to make the diagnosis.[32] This approach is as defensible as the diagnosis of measles during an epidemic of rubeola when confronted with a child manifesting cough, coryza, conjunctivitis, fever and Koplik's spots. In a non-endemic area the appearance and location of the lesions are not sufficient, particularly when the process is localized to a single portion of the respiratory tract. Some of the difficulties encountered in the differential diagnosis are illustrated in Case 3 and Case 4.

2. Histology.

The histology of the early and scarred end-stage of scleroma is generally agreed to be nonspecific.[1] It is in the florid granulomatous stage that specific changes have been described; these include: *a.* foamy, lacy cells often containing bacteria described by Mikulicz; *b.* predominately plasma cell exudate; *c.* Russell (Unna) bodies; *d.* proliferating connective tissue; *e.* hyalinization of vessels; and *f.* atrophy or hypertrophy of the covering epithelium.[2,12,14,37]

None of these features, however, are peculiar to or diagnostic of scleroma. Large macrophages containing bacteria similar to Mikulicz's cells are found in lepromatous leprosy, glanders, granuloma inguinale, and plague. A predominately plasma-cell exudate is a feature of many chronic inflammations. Russell's (Unna) bodies are degenerated plasma cells and are found in other inflammatory exudates composed primarily of plasma cells. Connective tissue proliferation and changes in the covering epithelium are repair processes that accompany chronic inflammation due to other known agents.

Following the concept of classifying histopathological processes in terms of injury-repair phenomena as fully developed by Forbus,[11] the above set of changes would characterize a chronic granulomatous inflammatory response to an agent of low virulence. The lack of necrosis and tubercle formation helps to separate it from some chronic inflammatory processes in which these features are prominent, but not from granulomas lacking these or other specific features whose etiology is well known.

3. Bacteriology.

Kouwenaar[21,22] used the recovery of *K. rhinoscleromatis* from the upper respiratory tract, with or without the clinical features of scleroma, for making the diagnosis. Many authorities would question this approach, as it equates scleroma with infestation of the respiratory passages by *K. rhinoscleromatis*. Some support for this view, however, is found in the study by Levine, Hoyt and Peterson[23] in which they were unable to recover *K. rhinoscleromatis* from normal noses or from the respiratory tracts of persons afflicted with conditions other than scleroma.

Whether recovery of *K. rhinoscleromatis* is essential to establishing the diagnosis as is insisted upon by some,[16,25] denied by others[22] and believed to be important (but not crucial) by still others,[27,29] is controversial. In the scarred end-stage or "burned-out" scleroma one would not expect to recover the organism.

4. Serology.

The value of the complement-fixation test using suspensions of killed or live *K. rhinoscleromatis* as antigen has been argued since this reaction was reported by Goldzieher and Neuber in 1909.[13] It can be considered supplemental evidence and possibly of value as a screening test. The techniques used in complement-fixation tests are subject to many variables and hence must be carefully conducted. To be reliable these tests require the services of an experienced immunologist.

Levine and Hoyt[24] reported that serum from scleroma patients fixed complement using a heat-killed suspension of *K. rhinoscleromatis* as antigen. They found that only 15 of 534 serums from patients with other assorted diseases gave positive reactions, and none of these were as strongly positive as those from scleroma patients. In a later communication Levine[26] noted that sera from scleroma patients also fixed complement with all the strains of *K. rhinoscleromatis* they had isolated, as well as with certain strains of *K. pneumonia* and *Aerobacter.*

The place of the skin test using killed *K. rhinoscleromatis* is even more equivocal.

SUMMARY.

Although scleroma has been a well-known clinical entity since 1870,[36] fundatmental observations on its etiology and epidemiology are still controversial. Favorable response to broad spectrum antibiotics has led to abandonment of radiation therapy as the treatment of choice. Disappearance of *Klebsiella rhinoscleromatis* from the respiratory passage with antibiotic therapy, often accompanied by dramatic clinical improvement in the florid stages, lends weight to the evidence that *K. rhinoscleromatis* is the etiologic agent of this entity. The possibility of a symbiotic virus-bacterial etiology, however, has not been excluded.

Our experience with scleroma in two long-term residents of Iowa and in two patients with scleroma-like lesions demonstrates some of the problems in differential diagnosis and

difficulties of establishing a diagnosis of scleroma in a non-endemic area.

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ADENOID REVISION.

Its Importance in the Treatment of Serous Otitis Media in Children.*

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Whereas recent developments in surgery of the middle ear for impaired hearing due to otosclerosis and chronic ear disease have been outstanding, they must not be allowed to overshadow the importance of preventive therapy for deafness. One of the most frequent causes of conductive hearing loss in children is serous otitis media associated with obstruction of the Eustachian tube. It is generally agreed that adequate adenoidectomy with aspiration myringotomy will usually result in relief of aural symptoms with restoration of normal function of the middle ear. In a series of 23 of my patients, however, who had had previous adenotonsillectomies, all experienced subsequent symptoms suggestive of fluid in the middle ear. Adenoid revision and aspiration myringotomy resulted in remarkable improvement of symptoms. It is the purpose of this study to point out that even with a history of adenotonsillectomy, examination for adenoid tissue (residual or regenerative) must be made in cases of serous otitis media, and any such tissue found must be adequately removed, and aspiration myringotomy should be done.

HISTORICAL DATA.

Many writers of early textbooks attributed fluid in the middle ear to adenoid obstruction to the Eustachian tube. Politzer,[1] in his chapter on "The Exudative Form of Middle Ear Catarrh," mentioned pressure of adenoid vegetations as an important cause of serous otitis media in children. He explained that the fluid was a bacteria-free transudate which

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formed in the tympanum as a result of the rarefaction of the air in the obstructed and impermeable tube. Perkins,[2] in noting that tenacious mucus is extremely difficult to remove by inflation, recommended that an incision be made in the membrana tympani so that the fluid might be forced out. He further noted that when adenoids were present, their removal usually resulted in improvement of hearing. Kerrison,[3] another early author, agreed that in nearly every case of tubal catarrh, the cause could be found in the nose or nasopharynx. On the other hand, Kopetzky[4] maintained that myringotomy did not improve hearing in catarrhal conditions but rather provided an inlet for infection.

Renewed interest in the problem was stimulated by the writings of Crowe and Burnam[5] who observed considerable improvement in hearing in children after irradiation of the nasopharynx. These authors recommended irradiation rather than a second operation for recurrent adenoid growth and advised aspiration myringotomy only when thick tenacious mucus was present. Modern thinking on the subject has been guided by the writings of Lawson,[6] Jones,[7] Robison,[8] Hoople and Blaisdell,[9] and Hantman.[10] Robison and Nicholas[11] elaborated in great detail upon the pathogenesis and treatment of catarrhal otitis media. They cited as causes of obstruction of the Eustachian tube, lymphoid tissue in the lateral pharyngeal recesses and salpingopharyngeal fold and pressure from the retropharyngeal nodes. Hoople[12] called attention to the diagnosis and treatment of the condition he termed "Otitis Media with Effusion" and noted that the fluid may be thin and serous or thick and seromucous. He described the technique of making a second, releasing opening, high and anterior in the drum to facilitate the suction of tenacious fluid. The use of plastic tubing inserted into, and allowed to remain in the myringotomy opening, has been reported by Armstrong.[13]

During the past decade many writers, including Hoopie,[13] Senturia and associates,[14] Suehs,[15] Theobald,[16] Singleton,[17] and Harcourt and Brown,[18] have reported the increasing frequency of serous otitis media. Cited as a possible cause for this rising

incidence by Senturia and associates[14] is inadequate antibiotic therapy, which leaves a smoldering subacute inflammation. This results in the formation of glycoprotein-secreting mucus and goblet cells. Finally, increased awareness and more diligent diagnosis must not be overlooked as factors in the increased incidence.

THE STUDY.

This study concerns 23 children, all of whom had had adenotonsillectomy and had subsequently had some disturbance of the middle ear which proved to be associated with obstruction of the Eustachian tube by nasopharyngeal lymphoid tissues. They ranged in age from three years to 14 years, with 15 of the 23 being age nine years or younger. Distribution by sex was equal, there being 12 females and 11 males. All of these cases were drawn from my own files and from those of my associates. The procedures done by us were performed in our private clinic over a period of one year. Clinical data of these patients are presented in Table I.

GENERAL CONSIDERATIONS.

Anatomy (Gross and Microscopic).

The nasopharynx, as described by Lederer,[19] is limited inferiorly by the upper surface of the soft palate. Anteriorly are the posterior choanae, separated by the nasal septum. On the lateral wall of the nasopharynx, behind the inferior concha, is the orifice of the Eustachian tube, the posterior lip of which presents a rounded cartilaginous prominence, the torus tubarius. A fold of mucous membrane, the salpingopharyngeal fold, which overlies a muscle of the same name, descends from the posterior-inferior part and gives the appearance of a hook. The pharyngeal recess, or Rosenmüller's fossa, is a vertical cleft located behind the orifice of the tube, extending under the petrous bone almost to the carotid canal. The roof of the nasopharynx, formed by the basioccipital and petrous bones and the body of the sphenoid bones, slopes into the posterior wall, which is formed by the prevertebral muscles and fascia overlying the atlas and axis. The pharyn-

geal tonsil or adenoids are located in this region and extend into the pharyngeal recess behind the orifice of the Eustachian tube.

Histologically, the adenoids consist of lymphoid tissue covered by pseudostratified ciliated columnar epithelium, which dips inward, forming vertical folds or crypts. The lymphatic tissue is separated from the surrounding structures by a thin fibrous coat.

The Eustachian tube, the tympanic cavity, the mastoid antrum, and the mastoid air cells join to form a continuous group of air-containing channels and spaces. In the adult, the Eustachian tube is approximately one and one-half inches long; it is composed of an inferomedial membrane-cartilaginous portion roughly one inch in length and a superolateral portion about one-half inch long. It constricts at the junction of two parts to form the isthmus. Under normal conditions it is opened regularly and periodically with each act of swallowing or yawning, by the movement of the tensor veli palatini muscle. The mucous membrane lining the tube is covered with ciliated columnar epithelium; it contains many seromucinous glands of mixed type near its pharyngeal end. The bony portion of the tube contains no glands.

The epithelium of the middle ear is of the simple cuboidal type, whereas that in the mastoid is of the simple squamous type. The nerves, blood vessels, and lymph channels traverse the submucosa. The walls of the vessel are extremely delicate. Neither the submucosa of the middle ear nor the mastoid normally contains any glands, but, according to Eggston and Wolff,[20] such glands may appear in the presence of infection. It is apparent from this anatomic structure that obstruction of the Eustachian tube renders conditions favorable for the transudation of fluid into the cavities of the middle ear and mastoid.

Pathogenesis and Sequelae.

The cause of fluid in the middle ear is thought to be blockage of the Eustachian tube, or failure of the tube to open properly to ventilate the middle ear. Singleton[17] contended

that closure of the Eustachian tube causes the air in the cavity of the middle ear and its extensions to be absorbed, and thus creates a vacuum in these cavities. As a result, dilation of the delicate blood vessels occurs, followed by transudation or exudation, which completely or partially fills the tympanic air spaces. Theobald[16] is of the opinion that once the fluid accumulates in the middle ear, a vicious cycle develops, the fluid causing edema of the tube to persist. This cycle can rarely be broken except by myringotomy.

Senturia and associates,[14] speculated that in early cases of acute rhinopharyngitis, viral or bacterial, obstruction of the Eustachian tube occurs, with effusion of a fluid high in protein and low in carbohydrate (mucus). In the absence of adequate antibody titers and administration of antibiotics, purulent otitis media develops. Spontaneous rupture of the tympanic membrane usually occurs, or myringotomy is performed to provide drainage of the purulent fluid. With inadequate antibiotics, the acute otologic symptoms may subside without drainage of the exudate from the tympanum, leaving a smoldering subacute inflammation. Metaplasia of the mucoperiosteal lining of the middle ear, with appearance of goblet cells and mucous glands, will occur with the secretion of glycoproteins. A mucopurulent fluid will then be present, and only adequate treatment to the nasopharynx, evacuation of the fluid, and control of respiratory infections will permit the mucous membrane to return to normal. Suehs[15] described the serous fluid as basically a transudate containing few inflammatory cells and usually having a low protein content, in contradistinction to the mucous fluid which is an exudate containing many inflammatory cells, mostly polymorphonuclears, usually with a high protein content.

If the tubal obstruction is neglected, irreversible changes may take place in the middle ear, such as fibrous fixation of the ossicles and necrosis of the long process of the incus; or, as Jordan[21] mentioned, primary acquired attic cholesteatoma may occur in cases of long-standing serous otitis media due to invagination and rupture of the pars flaccida with suppuration and ingrowth of epithelium.

The present series of 23 cases was divided into two subgroups. The children of Group 1 had the primary tonsillectomy and adenoidectomy a relatively short time before the secondary operation. These children had inadequate adenoid operation and needed aspiration myringotomy to remove fluid from the middle ear at that time. The children of Group 2 may or may not have had adequate operation, but fluid subsequently appeared in the middle ear with tubal obstruction due to adenoid growth. The etiologic factor in this group may have been allergy or recurrent bacterial or viral infection treated by antibiotics.

Clinical Data.

The parents of the first group of patients noted that their children had essentially the same symptoms as before the tonsillectomy and adenoidectomy: frequent attacks of earache, impaired hearing, mouth breathing, nasal obstruction, and frequent sore throats. Most of these children were seen three months to a year after their adenotonsillectomies.

The second group is composed generally of older children whose chief complaint was frequent attacks of earache and impaired hearing. It is my impression that children complain to their parents of ear pain more often than of hearing difficulty. Parents became aware of defective hearing in these children when they increased the volume of the television set, did not answer their calls, or became generally more irritable. Routine examinations at school and observations by school teachers revealed many hearing problems which had been undetected by parents.

Examination: Successful examination of children is enhanced considerably by maintaining the confidence of the children, by being gentle and avoiding hurting them. Often the canal must be cleansed of cerumen and epithelial debris before the drum can be seen. This is best accomplished by the use of spot suction, small wire loops, and grasping forceps. Most of the drums are dull, thickened, and retracted. The malleus handle is shortened and at times chalky white. One of the most important observations, made with the pneumatic

otoscope, is the sharply reduced, often almost absent, movement of the drum. With a cooperative child, study of the drum with magnification by the Ziess ear microscope is invaluable.

Examination of the nasopharynx is often difficult. In older children, a small postnasal mirror or a nasopharyngoscope can frequently be used. When the child will not permit this, it is almost always possible to use a mild shrinking nasal spray, and if the nasal septum does not obstruct it, the upper nasopharyngeal vault can be seen. I find that reflecting light from a clear glass light source (150 watt bulb) greatly facilitates visualization of the vault. A high ridge of adenoid tissue, as is often seen in patients who have had inadequate primary adenoidectomy, is usually easily visualized through the nasal vault. In some cases, it is impossible to visualize all areas of the nasopharyngeal vault unless the child has been anesthetized.

Hearing Test: In the younger children, the whisper test is usually possible. Theobald[16] pointed out that when the parents occluded one of the child's ears, the hearing loss was often dramatically demonstrated, since the parents could hear the whispered words very clearly, when the child could not.

A child with unilateral nerve-deafness may present a problem in differential diagnosis. A simple dramatic test involves masking the good ear by the Barany noise maker and determining whether the child can repeat words spoken with a loud voice into his affected ear. The fork test and audiometric studies should always be attempted even though they are unsatisfactory in some children, especially younger ones. When these tests can be carried out successfully, they characteristically indicate deafness of the conductive type.

Goodhill[22] has emphasized that confusion may occur because of the effect of the viscosity of the tympanic effusion on fenestral function, for, in some cases, audiologic examination may indicate a false nerve deafness because of a drop in bone conduction, which may disappear after removal of fluid from the middle ear.

Other problems to keep in mind in differential diagnosis of children with peripheral hearing losses are deafness or defective hearing, aphasia as a result of brain injury, emotional instability, and feeblemindedness. McHugh[23] has pointed out that, in general, children with peripheral hearing losses are usually consistent in their response or lack of response to sound stimuli.

Treatment: Treatment of all the children in the present series consisted in adequate adenoid revision and bilateral aspiration myringotomy. The following technique is recommended: Routine preoperative medical examination by the child's physician is advised. The parents are given written pre- and postoperative instructions. Vitamin K in the form of Synkavite ® is given for several days preceding the operation.

Appropriate oral dosages of Seconal ® and atropine sulfate (H) are used as preanesthetic medication. The anesthetic consists of gas induction with nitrous oxide or ethylene, followed by a switch to open ether by mask and then by insufflator hook when the mouth gag is in place. So far this has proven to be a safe method of anesthesia in my experience.

The nasopharyngeal vault is visualized by use of a metal tongue depressor and the Love soft palate retractor. A taut soft palate resulting from a previous adenotonsillectomy often makes visualization difficult.

The area is then explored digitally, and any adhesive bands from the posterior lip to the pharyngeal wall in the region of Rosenmüller's fossae are broken up. With the use of sharp Barnhill adenoid curettes, the lymphoid masses are removed first from the lateral pharyngeal recesses and then from the area of the central vault. It is important that sharp curettes be used, for when they are held in the vertical plane, the masses of lymphoid tissue can be neatly sliced off without digging into or tearing the deeper tissues. Primary bleeding usually ceases more rapidly, and secondary hemorrhage is much less likely with clean removal.

Any lymphoid masses seen along the salpingopharyngeal

folds or masses remaining on the posterior pharyngeal wall are removed with biting forceps. In some instances in which a high ridge of lymphoid tissue is present in the choanal region, I have, as Lake[24] recommended, inserted a suction tip through one nostril and placed the curette postnasally through the mouth by direct vision through the other nasal passage.

After the bleeding is controlled, the trachea is cleared of blood and secretions by intratracheal aspiration. Attention is then directed to the ears.

With the operator seated comfortably and with his gloves changed, the canal is cleansed with irrigation and spot suction by means of an electric otoscope. The drum is incised radially in the anterior-inferior quadrant, as suggested by Tremble,[25] because the radiating fibres are strongest in this area and tend to keep the incision open longer. Suction is then applied to this incision, and the fluid, if thin, can be removed easily. If the fluid is thick and tenacious, however, its removal may require a second, releasing incision higher and more anteriorly. The suction tip should never be placed inside the middle ear, where it may cause trauma to the mucosa of the medial wall. I recommend opening both ears, even though tests and examinations indicate that only one ear is involved, since in some instances, middle ear fluid has been found when it was not suspected.

REPORTS OF CASES.

Two representative cases from the two groups are presented.

Case 1: M. W., male child, age three years, was seen November 1959. The child was referred to me by a general practitioner, who had removed the tonsils and adenoids in June 1959. Nevertheless, the child continued to have attacks of "ear abscesses" for which he had been given numerous injections of penicillin as well as other antibiotics by mouth. The mother stated that the child was a "mouth breather" and had not seemed to hear well for several months before the adenotonsillectomy; his hearing had not improved afterwards. Whisper test indicated moderate hearing difficulty in both ears. Both drums appeared dull and retracted, and practically no movement of the drum was evident with the pneumatic otoscope. After nasal shrinkage, a large adenoid mass was seen postnasally on both sides. At operation, a large amount of lymphoid tissue was removed from the central vault and from both Rosenmüller's fossae. On aspiration myringotomy, both middle ear spaces were noted to be filled with a greyish, thick, tenacious fluid. On postoperative examination one week later, the child was alert and happy. The mother was obviously pleased with the child's improvement in hearing as well as in nasal

breathing. The ear canal was dry, and the drum incisions were well healed.

Case 2: J. A. T., an eight-year-old girl, was seen March 1960. The mother stated her child did not seem to hear well and had trouble breathing through her nose. These symptoms had been present before the child had her tonsils and adenoids removed ten months previously, and little improvement had been noticed after the operation. On examination, both drums were seen to be dull and retracted. The malleus handles were white and shortened. Audiometeric and fork tests revealed a moderate conductive type deafness in both ears. At operation two weeks later, a large mass of lymphoid tissue was curetted from the high central nasopharyngeal vault and moderate sized pieces were removed from the area of the right Rosenmüller's fossa. Thick, greyish, sticky fluid was removed from the right middle ear; the left middle ear contained considerably smaller amounts of the same type fluid. On postoperative examination one week later, the child's hearing was noticeably improved, with an average gain of 26 decibels in the right ear and 12 decibels in the left. The mother was pleased to report the disappearance of the "snoring noises" when the child was sleeping.

Case 3: D. A. B., an eight-year-old girl, was seen in May 1959. The mother stated that her child seemed to have poor hearing and had had recurrent attacks of earaches, which had been relieved by administration of antibiotics. At times, she noticed slight drainage from her left ear. The child had had an adenotonsillectomy when she was four years old. Examination revealed a scarred and retracted right drum. The left drum was moist and thickened, with no visible perforations. Audiograms revealed an average conductive loss of 20 decibels in the right ear and 30 decibels in the left. At operation on June 4, 1959, a high central mass of adenoid tissue and several pieces from both Rosenmüller's fossae were removed. Aspiration myringotomy yielded a small amount of the thick fluid from the right ear and large amounts of thick, greyish, sticky fluid from the left ear. On postoperative examination one week later, the child's hearing had improved. Audiograms yielded an average gain of 15 decibels in the right ear and 25 decibels in the left. Both drums were healed and dry, and the nasal passages were clear.

Case 4: K. B., a seven-year-old girl, was seen in December 1959 because of poor hearing, loud breathing noises at night, and frequent colds. She had had an adenotonsillectomy at the age of three years. Extremely thick, tenacious mucoid material was removed with difficulty from both nasal passages by spot suction. Obstructing adenoid tissue could be seen on both sides. Both drums were dull and retracted, with sluggish movement on pneumomassage. Audiograms revealed a decibel loss in the speech frequencies of 30-25-35-30 in the right ear and 25-25-30-30 in the left ear. Adenoid revision consisted of removing large pieces of tissue from the central area and the region of Rosenmüller's fossae by curettes. Aspiration myringotomy yielded a thick gluey material from both ears. One week later the hearing was improved, and the nasal spaces were clear. Audiograms at this time yielded an average gain of 20 decibels in the right ear and 20 decibels in the left ear.

COMMENT.

In the children in Group 1, in whom the primary adenoidectomy was obviously inadequate, the amount of lymphoid tissue removed was appreciably large; in fact, in several

instances the adenoid mass appeared to be untouched. In the children of Group 2, the removed lymphoid tissue was often small in amount but was usually found in the areas of Rosenmüller's fossae and along the salpingopharyngeal fold. The inadequate primary adenoidectomies were performed by general surgeons or practitioners whose habits include the use of overhead illumination and swipes at the adenoid mass with the La Force Adenotome. According to Blassingame,[26] at

TABLE I.

Results of Adenoid Revision and Aspiration Myringotomy in 23 Cases. Group 1.

Case	Sex	Age	Average Decibel Loss	Average Decibel Gain One Week Postoperative	Type of Fluid Removed	Recurrence to Date
			R—	R—	Seromucous	No
1.	M	3	L—	L—	Seromucous	No
			R-34	R-28	Seromucous	No
2.	F	8	L-32	L-28	Seromucous	No
			R-38	R-28	Seromucous	No
3.	M	7	L-34	L-24	Seromucous	No
			R-31	R-21	Seromucous	No
4.	F	8	L-28	L-20	Seromucous	No
			R-18	R-18	Seromucous	No
5.	M	6	L-28	L-20	Seromucous	No
			R-25	R-18	Seromucous	No
6.	M	7	L-28	L-20	Seromucous	No
			R-38	R-28	Seromucous	No
7.	F	6	L-36	L-28	Seromucous	No

least 50 per cent of the patients are operated upon by individuals unqualified by training to perform adenotonsillectomies. Some of the patients in Group 2 were, it is true, operated upon by competent specialists, but, as Hollender[27] has pointed out, hypertrophy of the lymphoid tissue will occur in the presence of recurrent infection.

When it is possible, the procedure of choice, I believe, is the technique of direct adenoidectomy, as described by Guggenheim[28] under visual control, with the use of the Love soft palate retractor. Alexander[29] has advocated the La Force Adenotome for the central adenoid mass and punch forceps for removal of lymphoid tissue in Rosenmüller's fossae. I

TABLE I.

Group 2.

Case	Sex	Age	Average Decibel Loss	Average Decibel Gain One Week Postoperative	Type of Fluid Removed	Recurrence to Date
			R-21	R-15	Serous	No
1.	F	8	L-31	L-22	Seromucous	No
			R-30	R-20	Seromucous	No
2.	F	7	L-28	L-20	Seromucous	No
			R-26	R-18	Seromucous	No
3.	F	13	L-24	L-16	Seromucous	No
			R-26	R-17	Seromucous	No
4.	F	12	L-24	L-16	Seromucous	No
			R-35	R-5	Serous	Yes
5.	M	14	L-40	L-10	Seromucous	Yes
			R-28	R-22	Seromucous	No
6.	F	12	L-30	L-22	Seromucous	No
			R-18	R-13	Serous	No
7.	M	9	L-16	L-12	Serous	No
			R-10	R-0	Dry	No
8.	M	14	L-41	L-11	Seromucous	Yes
			R-33	R-27	Seromucous	No
9.	F	9	L-35	L-28	Seromucous	No
			R-20	R-14	Seromucous	No
10.	M	7	L-20	L-14	Seromucous	No
			R-18	R-14	Serous	No
11.	M	12	L-20	L-15	Serous	No
			R-40	R-15	Seromucous	Yes
12.	M	13	L-10	L-5	Dry	No
			R-25	R-18	Seromucous	No
13.	F	8	L-25	L-18	Seromucous	No
			R-28	R-20	Seromucous	No
14.	F	7	L-26	L-20	Seromucous	No
			R-26	R-20	Seromucous	No
15.	M	10	L-30	L-25	Seromucous	No
			R-32	R-24	Seromucous	No
16.	F	9	L-30	L-24	Seromucous	No

favor the use of curettes throughout, as recommended by Houlihan.[30] In some cases the Jones[31] curettes are useful in removing adenoid tissue high in the nasopharynx. I have had no experience with the mirror visualization technique described by Grossan,[32] but I agree with his observations that in patients with recurrent aural disturbances one should look for a deep Rosenmüller's fossa containing considerable adenoid

tissue. I have not had occasion to use small biting forceps through the nasal passages, as described by Pederson,[33] to remove adenoid remnants.

In most of the cases herein reported, the fluid was a grey, thick, sticky, mucoid substance. Proud and Wehrs[34] noted that in early cases the fluid is thin and clear, whereas in long-standing cases the fluid is thick and tenacious and in some instances is of the consistency of jelly.

In one of the patients in the present series (Table I, Group 2, Case 5), adenoid revision and aspiration myringotomy were followed in one week by closure of the drum incision and

TABLE II.

Results of Adenotonsillectomy in 23 Children.

	Number of Patients Operated on by General Practitioner	Number of Patients Operated on by Specialist
Group 1. Symptoms before adenotonsillectomy not relieved by operation.	7	0
Group 2. Previous adenotonsillectomy with recurrence of symptoms.	10	6

recurrence of the serous middle ear fluid and deafness. For the next three months, he had repeated myringotomies with inflations in the office. He was then given a course of irradiation, and several weeks later both drums were tubed after aspiration of the serous fluid. The right tube was removed after one week, since the ear was dry and has remained so. The tube in the left drum has continued to drain for one month to date. The irradiation may or may not have relieved the right tubal obstruction, but it has not had any effect on the blockage of the left Eustachian tube. Meltzer[35] and Baron[36] believe that supplementary irradiation may be necessary to control postoperative recurrence of tubal closure. In my own practice I have discarded the use of the radium applicator completely and advise irradiation only in problem cases or in patients who have recurrences after adequate operation.

Results of operation in the patients studied were generally excellent; however, the response to allergic study and therapy should be considered in all children with recurrent serous otitis media, especially when the history and physical examination point to a definite coexisting allergic rhinitis. Reports by several writers indicate the frequency of an allergic basis for serous otitis media. Derlacki and Shambaugh[27] mentioned an allergic factor in 41 of 115 cases. Approximately 60 per cent of cases reviewed by Jones[28] over a six-month period were allergic in origin and were favorably influenced by allergic management. Jordan[21] classified 87 per cent of his series of 111 cases as allergic in origin.

Along with adequate operation, general corrective measures are applicable in all children with serous otitis media. Such measures include proper diet, with special attention to correction of overindulgence in carbohydrates; adequate rest; and avoidance of overexposure to heat or cold as, for example, from prolonged visits to swimming pools. I advise specific allergic studies and therapy, especially in children who have an obvious allergic rhinitis or who have repeated recurrence of fluid in the middle ear despite adequate adenoid operation and evacuation of the tympanic fluid.

Irreversible changes may occur in cases of long-standing serous otitis media. Bell[29] emphasized this in noting that the membrana tympani became flaccid and less tensile if the fluid were allowed to remain over a long period of time. A more serious change, the occurrence of attic cholesteatoma, has been discussed by Jordan.[21] The condition is produced by invagination and rupture of Shrapnel's membrane, caused by the middle ear vacuum, with resulting ulceration, suppuration, and epithelial ingrowth. Two cases in the present series demonstrate this sequence. In one boy of 14 years (Table I, Group 2, Case 8) with visual evidence of excessive adenoid tissue and undetermined cause of left conductive deafness due to the presence of a large aural polyp obscuring the drum, adenoid revision resulted in removal of a large central adenoid mass and appreciable lymphoid tissue from the left Rosenmüller's fossa. The aural polyp was removed; its origin had

been in the attic area associated with a thin retracted drum. It appeared that irreversible middle ear changes had probably occurred, and further major aural operation was, therefore, considered. The problem was explained to the parents, who agreed to allow the child to remain under observation. The ear became dry but the hearing, although improved, did not return to normal. Relief of the nasal obstruction and subsidence of the discharge in the left ear with some improvement in hearing caused the parents to believe that the child was cured. Ten months later, a foul discharge of the left ear and deafness appeared after the patient's first swim of the season. A left modified radical mastoidectomy was done, at which time an attic cholesteatoma was noted, with destruction of the distal half of the long process of the incus.

SUMMARY.

Adequate adenoid revision is of paramount importance in the treatment of conductive deafness due to serous otitis media in children who had previous adenotonsillectomy. In the present series of 23 cases of children with previous adenotonsillectomy but subsequent disturbances of the middle ear, most of the patients responded favorably to adenoid revision and aspiration myringotomy with infrequent recurrences. Under direct vision at operation, many of the primary adenoidectomies were seen to be inadequate. The site of the obstructing lymphoid tissue in cases with otologic symptoms was in Rosenmüller's fossae. Serous otitis media is a serious disease in children and, if allowed to persist, can lead to irreversible changes of the middle ear with resultant deafness and even danger to life.

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The next meetings of the American Society of Facial Plastic Surgery will be held on January 19, 1962, at the Hotel Elysee, 60 East 54th Street, New York, N. Y., and on March 3rd through March 5th at the Hotel Roosevelt, New Orleans, La. For further information address Samuel M. Bloom, M.D., Secretary, 123 East 83rd, New York 28, N. Y.

TRICLOBISONIUM CHLORIDE (TRIBURON*) IN EAR INFECTIONS.†

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External otitis is a dermatitis of the skin of the ear canal, and it has been treated with a wide variety of topical medications with more or less success. Despite the large number of topical medications available for use in external otitis, we are continually seeking new agents with a higher therapeutic index. The majority of these drugs currently on the market contain one or more antibiotics, hydrocortisone and a vehicle with acid pH. Unfortunately, the numerous strains resistant to many of the modern chemotherapeutic agents are on the increase[1]; therefore, every new drug which holds some promise in this field is welcome. Another special problem confronting physicians who treat external otitis is that of the B. proteus infections. The resistance of B. proteus to many of the modern antibiotics and antimicrobials is well known.[2,3] Encouraging results of preliminary investigation on the effectiveness of Triburon against B. proteus infections led us to investigate the present series of patients.[4]

Triburon is a newly synthesized wide spectrum antimicrobial agent (triclobisonium chloride)—a bisquaternary diamine derived from betaionone. Triburon is not an antibiotic, sulfonamide or nitrofuran. As a topical agent it has been used with success in a variety of dermatologic and surgical conditions.[5-9]

It has been shown to exert a high bacteriostatic and moderate bactericidal activity against a wide range of gram posi-

*Triburon supplied by Hoffmann-La Roche Inc., Nutley, N. J.

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tive and gram negative organisms *in vitro*.[10] In clinical trials, Triburon had a marked activity against most of the common skin pathogens, including Staphylococcus aureus, Staphylococcus albus, Streptococcus pyogenes and, of particular interest to otolaryngologists, B. proteus.

Reports on Triburon have shown it to be both effective and safe. It does not produce sensitization or primary irritation, and is non-toxic, when used in topical application.

TABLE I.

Results of Treatment with Triburon of B. Proteus Infections.

Indication	No. of Patients	Organism Cultured	Clinical Results	
			Satisfactory	Unsatisfactory
Acute diffuse external otitis	8	B. proteus Hem. staph.	8	0
Chronic diffuse external otitis	5	B. proteus Pseudomonas	4	1
Chronic otitis media	32	B. proteus Pseudomonas	20	12
Post-surgical infections	7	B. proteus Pseudomonas	3	4
Total	52		35	17

MATERIALS AND METHODS.

A total of 92 patients in two groups were treated with Triburon plus hydrocortisone at the Out-Patient Department of University Hospital, Baltimore, Maryland, for various ear infections, but predominantly for acute external otitis and chronic otitis media. The first group consisted of ear infections where B. proteus was the prominent organism with other bacteria present only as contaminants. In the second group, no proteus infection was present, but common ear pathogens prevailed.

The first group consisted of 52 patients, ranging in age from three months to 56 years, in whom B. proteus was the dominant organism (see Table I). There were 17 children

in this group. The conditions treated in order of frequency included chronic otitis media, acute diffuse external otitis, chronic diffuse external otitis and post-surgical infections. The majority of these patients were treated after the condition had been present for from one month to several years. Nineteen patients had had previous medications consisting of other otic solutions, containing primarily various antibiotics; also systemic antibiotics, all of which failed to resolve the persistent discharge. Triburon with hydrocortisone was

TABLE II.

Results of Treatment with Triburon of Common Ear Infections Involving Pathogens Other Than B. Proteus.

Indication	No. of Patients	Organism Cultured	Clinical Results	
			Satisfactory	Unsatisfactory
Acute diffuse external otitis	23	Hem. staph. aureus Pseudomonas Beta Hem. strep. Enterococcus E. coli	20	3
Chronic diffuse external otitis	4	Hem. staph. Pseudomonas Enterococcus	3	1
Chronic otitis media	10	Pseudomonas Hem. staph. E. coli	7	3
Acute otitis media	3*	Hem. staph.	3	0
Total	40		33	7

*Triburon used in combination with Madribon.

the only therapeutic agent used in the majority of these cases, except for six children under age three where the seriousness of the condition warranted the use of systemic antibiotics in combination with the topical preparation. Seven patients had previous surgical procedures: four of them radical mastoidectomy; two modified radical mastoidectomy, and one tympanoplasty two to four months prior to treatment.

Culture studies showed that B. proteus was the predominant

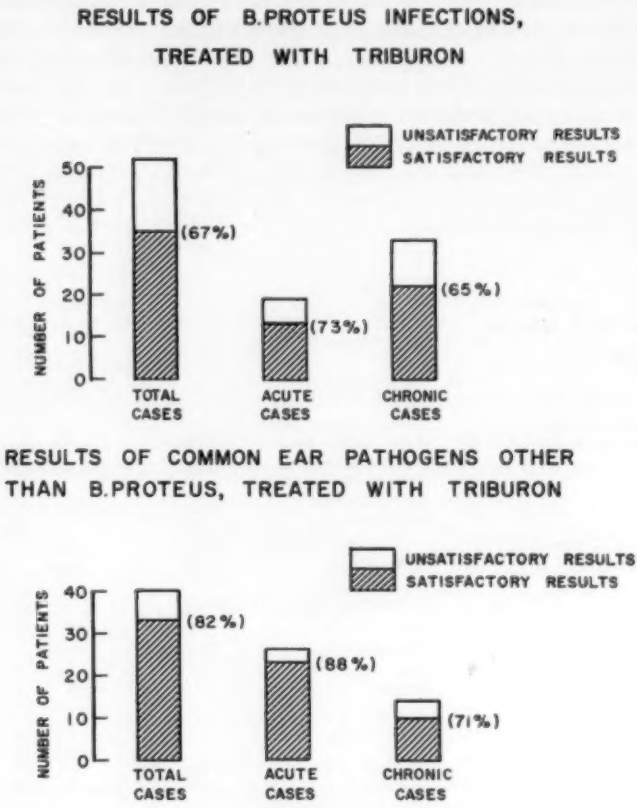

Fig. 1.

organism in all of these cases, with Pseudomonas and other bacteria present only in small numbers as contaminants.

In the second group, there were 40 patients, ranging in age from two months to 62 years, who received Triburon with hydrocortisone for various ear infections caused by common ear pathogens, frequently in combination but without the presence of B. proteus (see Table II). These conditions

treated in order of frequency were acute diffuse external otitis, chronic otitis media, and chronic diffuse external otitis.

Culture studies performed on all of these patients in the second group showed a wide variety of ear pathogens. There were in decreasing frequency: Staphylococcus aureus, Pseudomonas, Beta-hemolytic strep., E. coli, enterococcus. These organisms were present frequently in combinations, but none of them contained B. proteus. Triburon with hydrocortisone was used in the majority of these cases, except for three cases of severe acute diffuse external otitis involving infants, where Madribon was administered systemically, along with topical application of Triburon. An average of three drops of Triburon Otic Solution was instilled in the ear three times a day (Triburon—0.1 per cent, hydrocortisone—1.6 per cent in a special propylene glycol base).

RESULTS.

In group one, where the predominant organism was B. proteus, results were satisfactory in 67 per cent of the cases, 35 out of 52. Among the 15 acute cases, 11 or almost 73 per cent responded satisfactorily. A somewhat lower response was found for the chronic cases where 24 out of 37 cases were counted as satisfactory. Of the 32 cases of chronic otitis media, repeated cultures showed that in eight cases Pseudomonas became the prominent organism, and six of these cases did not respond favorably to the treatment. The average period of treatment was seven to 14 days in this group. In the children (six) who received antibiotics, the required period to clear up the infection was not considerably shorter. Results were unsatisfactory in 17 patients.

In the second group where B. proteus was not present in the culture, the result was satisfactory in 33 out of 40 cases, *i.e.*, 82 per cent. This group consisted primarily of acute ear infections, mainly acute external otitis. The over-all results in this second group were more favorable, thus confirming the previously published reports concerning the wide microbicidal effects of Triburon. The majority of cultures obtained showed the presence of Staphylococcus aureus, but Beta-

hemolytic streptococcus, Pseudomonas, and E. coli were also isolated. Two patients complained of transient burning sensation on initial application but without evidence of subsequent irritation. Treatment was successful in an average of six to ten days. Results were unsatisfactory in seven patients.

COMMENT.

Triburon plus hydrocortisone in a special propylene glycol base was found to be a valuable non-antibiotic synthetic compound for the elimination of various ear infections, when topical medications were indicated. Its effectiveness against common ear pathogens was very good. Triburon was also effective in a relatively high per cent of our study in eliminating ear infections caused by B. proteus, which we feel makes it high on the list in combating this sometimes quite resistant organism.

SUMMARY.

A new antimicrobial compound, Triburon with hydrocortisone in an otic solution, was used as topical therapy for 92 patients with various ear infections. Group one consisted of 52 patients where B. proteus was the predominant organism. Treatment with Triburon Otic Solution alone for an average of 7 to 14 days successfully cleared the infection in 67 per cent of the cases. An average of three drops of Triburon was given three times a day. The additional administration of systemic antibiotics to six patients in combination with topical Triburon did not seem to shorten the course of the treatment.

The second group consisted of 40 patients. Culture studies showed various common ear pathogens, but no B. proteus in this group. The results were satisfactory in 82 per cent of the cases, after an average length of treatment of six to ten days.

The majority of cases which did not respond favorably to Triburon Otic Solution consisted of chronic ear infections, especially chronic otitis media. Except for two patients who

complained of transient burning sensation, Triburon with hydrocortisone was tolerated unusually well. No systemic side reactions were encountered.

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COURSE IN OTOLARYNGOLOGY.

A medical continuation course, Otolaryngology for Specialists, will be presented at the Center for Continuation Study, University of Minnesota, Minneapolis, Minnesota, January 25-27, 1962. For further information address W. Albert Sullivan, Jr., M.D., Director, 1342 Mayo Memorial, Minneapolis 14, Minn.

A SIMPLE INSTRUMENT FOR INSERTION OF POLYETHYLENE TUBES.

FRANK V. MANIERI, M.D.,

Baltimore, Maryland.

After experiencing extreme difficulty in positioning a polyethylene strut, I decided there must be an easier way, and designed a simple instrument with a new mechanical principle. The idea was presented to Mr. Fred Geiwitz, of Murray-Baumgartner Surgical Supply Company, Inc., and the present instrument was made. So far it has been in use for over a year and tried by a number of otologists, all of whom liked the principle and instrument. It is upon their suggestion and that of an instrument maker's representative, who had a duplicate made and sent to the main office, that I am reporting on this. I hope it may facilitate the surgery for others.

Placement of the polyethylene strut is now extremely easy. No longer is there the possibility of the tube's sticking to the usual alligator-type carrier. It can be positioned accurately, as close to the lenticular process as may be desired.

The present instrument consists simply of a piece of tight 18 gauge stainless steel tubing, 3⅜ inches long, through which a stylet is passed. The stylet protrudes 2 mm. from the end of the tubing. The distal 1 mm. is tapered almost to a point. This is important, otherwise the polyethylene strut will spring off. At the other end is a spring handle 2 inches long to which the tubing is soldered. The stylet in turn is soldered to the other end of the spring. On squeezing the spring handle together, the stylet is drawn back and releases the strut. Polyethylene tubes can be accommodated.

This instrument may also be used for inserting polyethylene tubes in other conditions such as serous otitis. A larger tube and stylet can be used to accommodate larger polyethylene tubes for other procedures.

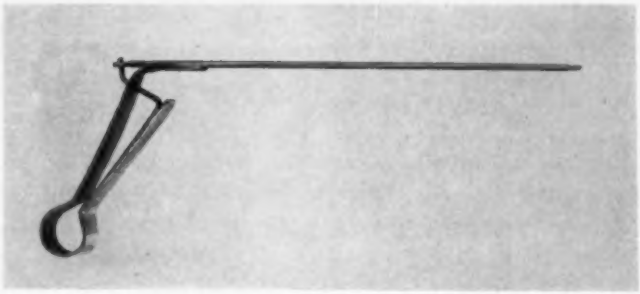

Fig. 1.

SUMMARY.

A new instrument with a new mechanical principle for easy insertion of polyethylene struts in otosclerosis is presented. This instrument may be purchased from the manufacturer, Murray-Baumgartner Surgical Supply Company, Inc., 1421 Maryland Avenue, Baltimore 1, Md.

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY, UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from April 2 through 14, 1962, under the direction of Paul H. Holinger, M.D.

Registration will be limited to 15 physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

IN MEMORIAM.

CHARLES DECATUR BLASSINGAME, M.D.,

1884-1961.

Charles Decatur Blassingame was born in Booneville, Mississippi, December 22, 1884, and died in Memphis, Tennessee, June 2, 1961.

He received his high school education at Chalybeate High School in Mississippi. He graduated from Mississippi College in 1911 (Ph.B. cum laude). He was principal of the Tallahassee County High School in Cacill, Mississippi, the following year. His first two years of medicine were spent at the University of Mississippi. His degree of Doctor of Medicine was obtained from Vanderbilt University, Nashville, in 1916.

Dr. Blassingame served one year in general practice in Itawanibi County, Mississippi, and came to Memphis in 1917. He joined his cousin, Dr. J. F. Hill, Robert Fagan and W. Likely Simpson in practice. That same year he volunteered for the army and was assigned to the Ear, Nose and Throat Division of the Evacuation Hospital No. 49 where he later became Head of Otolaryngology. He remained at this Base Hospital, Joinville, France, until the Armistice, when he was discharged as a Captain. He accepted an appointment as Assistant House Surgeon at the Golden Square Throat Hospital in London, where he remained for one year, and where he made many friends.

On returning to the States, Dr. Blassingame joined the staff of the Baptist Hospital and was made assistant professor at the University of Tennessee. In 1923 he became a Fellow of the American Academy of Ophthalmology and Otolaryngology and was certified by the American Board in 1925. He was elected to Fellowship in the American College of Surgeons in 1926. In 1928 he was elected to Fellowship in the American Laryngological, Rhinological and Otological Society, Inc., serving as vice president of its Southern Section

in 1937. He became a Fellow of the American Laryngological Association in 1938 (Vice President 1958), the American Otological Society, in 1942 and the American Broncho-Esophagological Association, in 1946.

He served as Vice President of the Baptist Hospital Staff, Secretary of the Memphis Eye, Ear, Nose and Throat Society, President of the Tennessee Academy of Ophthalmology and Otolaryngology and held many other offices. He was chief and head of the Department of Otolaryngology, University of Tennessee Medical School from 1948 to 1954. Dr. Blassingame's contributions to literature were numerous. He was an avid reader and enjoyed writing. At the time of his death he was preparing a treatise on a phase of the treatment of sinusitis.

Dr. Blassingame was one of seven children. He lost his father, William Scott Blassingame, and a senior brother during his high school days. His mother, Elizabeth Patterson Blassingame, accepted the responsibility of the family and was justly proud of her children. In 1934 Charlie married Glenn Johnson, the daughter of the pioneer in plastic surgery in the South. They remained a very devoted couple and their friends, in all walks of life, were numerous.

About 19 years ago Charlie and Glenn purchased a cottage on a high bluff in the Ozarks at Forsyth, Missouri, overlooking Bull Shoals Lake. Every Summer they would retreat to the Ozarks and enjoy the natives and their friends at "Chiggervilla". He was considering retiring from his active practice in Memphis and establishing a clinic in the Ozarks where he could look after the needy patients.

Dr. Blassingame's hobbies were his friends, the University and golf. He was an ardent golfer and seldom traveled without his golfing equipment. He was always interested in teaching, gave freely of his time to the University and was still active after being made professor emeritus. His library, office equipment and instruments were bequeathed to the Department of Otolaryngology, University of Tennessee.

His friends always came first. Nothing was too important to come before a friend, and as one of them stated, "He meant so much to so many families."

He was on the Board of Stewards of the St. John's Methodist Church and a faithful member. He is survived by his widow, three brothers and a sister.

S. H. S.

THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, West Virginia, on April 23-25, 1962.

The guest speakers for Ophthalmology are:

Dr. Ramon Castroviejo, New York City, New York
Dr. Frank D. Costenbader, Washington, D. C.

The guest speakers for Otorhinolaryngology are:

Dr. Kelvin A. Kasper, Philadelphia, Pennsylvania
Dr. Fred R. Guilford, Houston, Texas

In addition, arrangements have been made with Mr. Philip Salvatori of Obrig Laboratories to discuss and show techniques of contact lens fitting.

A registration fee of $25 for associate members will cover all the social and scientific sessions.

For additional information, please contact the secretary, Dr. Worthy W. McKinney, 109 East Main Street, Beckley, West Virginia.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Lawrence R. Boies, Milwaukee, Wisc.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Las Vegas, Nev., November 3-9, 1962.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

President: Dr. J. J. Longacre, 1503 Carew Tower, Cincinnati, O.
President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting:

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Palmer House, Chicago, Ill., Oct. 28-Nov. 1, 1962.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Alden H. Miller, 500 S. Lucas Ave., Los Angeles, Calif.
Vice-President: Dr. Herbert W. Schmidt, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Daniel C. Baker, Jr., 903 Park Ave., New York 21, N. Y.
Treasurer: Dr. Charles M. Norris, 3401 No. Broad St., Philadelphia 40, Pa.
Meeting: Sheraton-Dallas Hotel, Dallas, Tex., May 1-2, 1962 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Dean M. Lierle, Iowa City, Ia.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Francis W. Davison, Danville, Pa.
Meeting: Sheraton-Dallas Hotel, Dallas, Tex., May 4-5, 1962.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. John R. Lindsay, Chicago, Ill.
President-Elect: Dr. Howard P. House, 2122 W. Third St., Los Angeles 57, Calif.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Sheraton-Dallas Hotel, Dallas, Tex., May 1-2-3, 1962 (mornings only).

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Howard P. House, Los Angeles, Calif.
Vice-Chairman: Dr. Joseph L. Goldman, New York, N. Y.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Paul H. Holinger, Chicago, Ill.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, O.
Meeting: Chicago, Ill., June 11-15, 1962.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Lawrence R. Boies.
Secretary-Treasurer: Dr. James A. Moore, New York City, N. Y.
Annual Meeting: Sheraton-Dallas Hotel, Dallas, Tex., April 29-30, 1962.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worcester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ivan W. Philpott, 1801 High St., Denver, Colo.
Secretary: Dr. Robert M. Hansen, 2210 Lloyd Center, Portland 12, Ore.
Annual Clinical Session:

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY.

President: Dr. John J. Conley, New York, N. Y.
Vice-President: Dr. Joseph H. Ogura, St. Louis, Mo.
Treasurer: Dr. F. Johnson Putney, Philadelphia, Pa.
Secretary: Dr. George A. Sisson, Syracuse, N. Y.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Vice-President: Dr. John T. Dickinson, Mercy Hospital, Pittsburgh 19, Pa.
Secretary: Dr. Samuel M. Bloom, 123 East 83rd St., New York 28, N. Y.
Treasurer: Dr. Joseph C. Miceli, 879 Glenmore Ave., Brooklyn, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Leland H. Prewitt, Ottumwa, Ia.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Alberto Gallo.
First Secretary: Dr. Alfredo Martinelli.
Second Secretary: Dr. Guedes de Melo Neto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. Antonio de Almeida, Dr. Gabriel Pôrto, and Dr. Roberto Franco do Amaral.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Fernand Montreuil, 1123 St. Joseph Blvd. East, Montreal, Quebec.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. E. Hartenbower, 203 N. Main St., Bloomington, Ill.
President-Elect: Dr. Edgar T. Blair, Springfield, Ill.
Vice-President: Dr. G. LeRoy Porter, Urbana, Ill.
Delegate at Large: Dr. S. G. Baldwin, Danville, Ill.
Secretary-Treasurer: Dr. C. L. Pannabecker, Peoria, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. George Woodruff, Woodruff Clinic, Joliet, Ill.
Vice-President: Dr. Linden Wallner, 122 So. Michigan, Chicago, Ill.
Secretary-Treasurer: Dr. Robert Lewy, 25 East Washington St., Chicago 2, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. James A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. P. W. Rhyne, Albany, Ga.
Vice-President: Dr. C. L. Pennington, Macon, Ga.
Secretary: Dr. James T. King, 516 Baptist Professional Bldg., Atlanta 12, Ga.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Paul H. Holinger, 700 N. Michigan Avenue, Chicago 11, Ill.
Secretary: Dr. Charles M. Norris, 3401 N. Broad Street, Philadelphia 40, Pa.
Meeting: 1963.

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Carl E. Granberry.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., April 13-14, 1962.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairman in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Petrarca 332-1, Mexico 5, D. F.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

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First Vice-President: Dr. John A. McNeill, St. Paul, Minn.
Second Vice-President: Dr. Benjamin Bofenkamp, Minneapolis, Minn.
Secretary-Treasurer: Dr. Richard O. Leavenworth, Jr., St. Louis Park, Minn.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. L. B. W. Jongkees, Koninginneweg 157, Amsterdam.
Secretary: Dr. W. H. Struben, Essenlaan 72, Rotterdam.
Treasurer: Dr. A. G. Boon, Apollolaan 48, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George T. Noel, Kannapolis, N. C.
Vice-President: Dr. J. C. Bradley, Weaverville, N. C.
Secretary: Dr. John S. Gordon, 1350 South Kings Drive, Charlotte, N. C.

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. George C. Saunders, 644 Medical Arts Bldg., Portland 5, Ore.
Secretary-Treasurer: Dr. Donald C. Mettler, 1216 S. W. Yamhill St., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Aero Club, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Victor Goodhill, 4759 Hollywood Blvd., Los Angeles, Calif.
Vice-President: Dr. Sam Rosen, 101 E. 73rd, New York, N. Y.
Secretary-Treasurer: Dr. Ray E. Jordan, 3515 Fifth Ave., Pittsburgh, Pa.
Meeting: Las Vegas, Nev., November 2, 1962.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118-9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Plino de Mattos Barretto, Sao Paulo, Brazil.
Acting Executive Secretary: Dr. Charles M. Norris, 3401 No. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Eighth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, Puerto Azul Club, Valencia, Venezuela, S. A., February 25th to March 1st, 1962.

THE PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin H. Shuster, Philadelphia, Pa.
President-Elect: Dr. Norbert F. Alberstadt, Erie, Pa.
Secretary: Dr. Daniel S. DeStio, Pittsburgh, Pa.
Treasurer: Dr. Bruce A. Grove, York, Pa.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Joseph P. Atkins.
Vice-President: Dr. Louis E. Silcox.
Secretary: Dr. Charles M. Norris.
Treasurer: Dr. William A. Lell.

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Cesar F. Villafuerte.
Vice-President: Dr. Napoleon C. Ejercito.
Secretary-Treasurer: Dr. Eusebio E. Llamas.
Directors: Dr. Antonio L. Roxas and Dr. Armando T. Chiong.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Vice-President: Dr. Carson S. Demling, 513 Jenkins Bldg., Pittsburgh 22, Pa.
Secretary-Treasurer: Dr. Clyde B. Lamp, 8101 Jenkins Arcade, Pittsburgh 22, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

ROYAL SOCIETY OF MEDICINE.
SECTION OF LARYNGOLOGY.

President: N. Asherson, F.R.C.S.

Vice-Presidents: E. J. Gilroy Glass, F.R.C.S.
Kennedy Hunter, F.R.C.S.
Robert Marshall, F.R.C.S.

Honorary Secretaries: Alun B. Thomas, F.R.C.S.
Leslie Salmon, M.B.E., M.S.

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Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SEVENTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

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Sede: Avenida da Liberdade, 65, 1°, Lisboa-Portugal.

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Secretary-Treasurer: Lt. Col. Donald J. Joseph, USA, MC, Madigan General Hospital, Tacoma, Wash.
Meeting: Concurrent with the Academy of Ophthalmology and Otolaryngology, Las Vegas, Nev., November 3-9, 1962.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Secretary: Dr. Albert C. Esposito, Huntington, W. Va.
Meeting: Miami Beach, Fla., November 12-17, 1962.

SOUTH WESTERN LARYNGOLOGICAL ASSOCIATION.
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WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Vice-President: Dr. James T. Spencer, Charleston, W. Va.
Secretary-Treasurer: Dr. Worthy W. McKinney, Beckley, W. Va.
Meeting: Greenbrier Hotel, April 22-25, 1962.